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LECTURES

ON

DISEASES OF THE PLEURA.*

By CECIL WALL, M.D., F.R.C.P.,

Physician to the London Hospital.

LECTURE I.

I HAVE taken as the subject of my summer course of lectures the diseases of the pleura.

The subject is a large one, and I propose to deal but briefly with the familiar points, and to draw your attention to certain matters which are less well known, or have recently been the objects for speculation and research.

Anatomical.—The pleura in health is a smooth serous membrane, of which part invests the lungs completely, extending into the fissures nearly to the hilum, and part lines the chest-wall, the mediastinum and the diaphragm. The relations of the pleura are of great practical importance because of the gravity of the result when inflammation or other disease spreads to it from a neighbouring viscus.

The frequency of the relation between pulmonary disease and pleurisy is so well known and the connection between injuries and diseases of the chest-wall and pleurisy is so simple of explanation that we need not stay to consider them.

I would remind you of the close relation between the parietal pleura and the intercostal nerves. Inflammation of the pleura may at times result in irritation of the intercostal nerves, and the resulting painful sensations may be referred to the peripheral distribution of those nerves. To this matter I shall have occasion to refer later when we discuss the simulation of abdominal disease by morbid intra-thoracic changes.

The relation of the parietal pleura to the bodies of the vertebræ is of importance when those

* Delivered at the London Hospital.

bones are diseased. Recently there was in Yarrow Ward under my care a woman with compression paraplegia, due to caries of the spine in the dorsal region. A small dose of tuberculin was given for diagnostic purposes; this was followed almost at once by a general febrile reaction and a pleural effusion which took three weeks to subside. It seems very probable that this is an example of pleurisy secondary to disease of the vertebræ. Possibly the inequality of the pupils so frequently found in cases of pleurisy may be explained by the fact that the rami communicantes and the sympathetic lie in contact with the external surface of the parietal pleura.

As the pleura passes from the vertebræ to the root of the lung its connections are of practical importance; disease of the lymph-glands, bronchi or œsophagus may spread to it; it seems certain that tuberculosis of the bronchial glands is a common cause of pleurisy with or without effusion. These three specimens show the mode of spread.

The first specimen was from a boy, æt. 5 months, admitted to the hospital in 1906 under Dr. Lewis Smith (Museum Catalogue number 1701A).

On admission the diagnosis of tuberculous glands of the neck and peritonitis was made.

At the autopsy the cervical and bronchial glands were found to be enlarged and caseous.

The specimen shows that the tuberculous process spread to the pleura along the lymphatics; the two layers of the pleura were adherent at the site of the disease and no effusion was present. The lung substance was free from tuberculosis.

The second specimen was taken from a body in the post-mortem room on August 9th. The patient was admitted with chronic parenchymatous nephritis, had ten uræmic fits, and died in coma.

At the post-mortem examination caseous tuberculous glands were found in various parts of the body; there was a small pleural effusion on the left side; miliary tubercles were seen spreading along the subpleural lymphatics from a caseous bronchial gland. There were submiliary tubercles throughout the lungs. The kidneys showed the changes of chronic parenchymatous nephritis.

The third specimen is a lung in which the subpleural lymphatics are filled with carcinomatous cells spreading from a secondary carcinomatous gland at the root of the lung.

The pleuræ are also in contact with the external layer of the pericardium, and this relation at times offers an explanation of disease. Above the first rib the pleura is in relation with the contents of the posterior triangle of the neck, and extension of disease, especially from the lymphatic glands, sometimes occurs. At the lower part of the thorax the pleura covers the diaphragm and may be implicated in disease spreading through this muscle from the abdomen.

The pleural cavity in health is purely potential, two pleural surfaces always being in contact. Alteration in the size of the lungs during respiration is permitted by certain so-called sinuses of the pleura.

The *costo-mediastinal pleural sinus* is the complementary space between the sternal line of reflexion and the anterior border of the lung; on the right side this is completely filled during inspiration by the expanded lung. On the left side, however, the part of the sinus corresponding with the area of superficial cardiac dulness is present even during inspiration; this is sometimes termed the *pericardio-sternal sinus*. It is filled by the lung when there is advanced emphysema.

The *costo-phrenic pleural sinus* is the potential space between the lower border of the lung and the diaphragmatic line of pleural reflexion. It is never completely occupied by the lung even during deep inspiration, but is much lessened in emphysema.

The depth of this sinus varies in individuals; measured in the living by percussion the lung is found to descend during inspiration about 1 in. in the right mid-clavicular line, about $2\frac{1}{2}$ to 3 in. in the mid-axillary lines, and about 2 in. in the right posterior scapular line.

The left pleura is reflected posteriorly from the eleventh and twelve ribs at a slightly lower level than that on the right side. In the expiratory position, however, the lower border of the right lung is on a level an inch or an inch and a half above that of the left lung. For this reason the right costo-phrenic sinus behind is considerably deeper than the left. In percussion of the normal chest, resonance extends lower on the left side behind than on the right; a knowledge of this fact is of importance in the diagnosis of some diseases of the pleura.

In the axillæ during inspiration the diaphragm, as it were, is peeled off the inner surface of the

chest-wall, and the lung descends into the costo-phrenic sinus. Litten pointed out that by means of oblique illumination a moving shadow could be demonstrated in the axillæ during respiration; he showed that if the costo-phrenic sinus were full of fluid that this moving shadow could not be seen. The presence of adhesions preventing the entry of the lung into the sinus has a similar effect, so that the sign by itself cannot be taken to be pathognomonic of pleural effusion. When adhesions are present on the right side careful percussion may show that the lung does not descend into the sinus during inspiration.

On the left side, in the area known as Traube's semi-lunar space, the costo-phrenic sinus is superficial to the stomach, and a resonant note is obtained over it even when it is not filled by lung. When this part of the sinus is filled with fluid a dull percussion note may be obtained.

A knowledge of the arrangement of the costo-phrenic sinus is of importance, not only in the diagnosis of diseases of the pleura, but also in deciding site of such surgical procedures as aspiration of pleura, or pericardial effusion and resection of ribs for empyema, or as a preliminary measure before draining a subphrenic abscess.

As the parietal pleura investing the vertebræ passes forward to cover the mediastinum it forms a sinus known as the vetebro-mediastinal pleural sinus. On the left side this is seldom deep owing to the presence of the aorta; on the right side a deep recess may cross the mid-line below the level of the pulmonary roots, and bring the pleura into relation with the right side of the œsophagus. When there is a pleural effusion it is supposed that the sinus gets filled; the damping of the vibrations of the bodies of the vertebræ by the fluid is taken to be the explanation of the para-vertebral triangle of dulness on the side opposite to the effusion, which was described by Grocco and bears his name.

Ætiology.—It is usually said that pleurisy may be either primary or secondary. Primary pleurisy in the strict sense must be of great rarity, but pleurisy in association with such general diseases as rheumatism, pyæmia or Bright's disease is not very uncommon, and in so far as it is not the result of direct extension of inflammation may be considered to be primary.

Secondary pleurisy results from the spread of inflammation from some neighbouring structure. Diseases of the lung which reach the surface are responsible for a very large number of cases of pleurisy; pneumonia, tuberculosis, abscess, gangrene, infarcts, new growths, are examples.

Pleurisy may have a starting-point in the chest-wall as a result of injury, or from disease, *e. g.* tuberculous periostitis, syphilitic disease, carcinoma of the breast, malignant and tuberculous glands in the posterior triangle of the neck, etc.

Disease of the contents of the mediastina not infrequently leads to pleurisy; the glands at the root of the lungs lie in close proximity to the pleura, and being frequently the site of inflammation, are often primarily responsible for an attack of pleurisy.

The association of pericarditis with pleurisy is frequent; less commonly disease of the trachea, bronchi and œsophagus spreads to the pleura. Finally abdominal disease, such as subphrenic abscess, may extend through the diaphragm and excite pleurisy.

With this formidable list of possible routes of infection it is not surprising that numerous organisms have been found present in different cases. Apart from pleurisy due to malignant disease or infarction, it is probable that all cases are due to an infection and are truly inflammatory: the use of the term "pleuritis" to draw attention to this fact is perhaps advisable.

If cold plays any part in the production of pleurisy it is probably by lowering the local resistance against infection.

The tubercle bacillus is such an exceedingly common and important excitant of pleuritis that clinically it is convenient to distinguish the two groups, tuberculous and non-tuberculous. Of organisms other than the tubercle bacillus which have been found to cause pleuritis, the pneumococcus is the commonest; streptococci, staphylococci, *Coli bacilli*, *B. typhosus*, saprophytic organisms have been found. The hypothetical *Micrococcus rheumaticus* has also been isolated. It is frequently found that the infection is mixed.

To the morbid anatomy of pleurisy I need allude but briefly. As in the inflammation of other serous membranes, the first stage recognisable by the naked eye is loss of lustre, said to be brought about by plugging of the stomata and lymphatics with fibrin and corpuscles. Shortly after this a

fibrinous exudate appears on the surface; in its meshes are entangled leucocytes and red corpuscles.

At this stage the inflammation is still said to be "dry," and may resolve; if this occurs it is probable that in most, if not all cases, the exudate becomes vascularised and transformed into connective tissue. In most cases of pleurisy both of two opposed surfaces become inflamed, and after resolution remain united by the connective tissue. Sometimes, however, resolution may occur without subsequent adhesion.

If resolution does not occur in the dry stage a fluid exudate collects in the pleural cavity. This exudate may be serous or purulent. The constitution of the exudate will come up for consideration later.

Symptomatology.—The onset of pleurisy is usually somewhat sudden: the patient, without any previous period of malaise, complains of pain in the side, increased by the respiratory movements, and of a dry cough. As a rule there is some pyrexia, with a sensation of chilliness, or even a rigor.

Should the pleurisy develop in association with some other disease such as pneumonia or pulmonary tuberculosis, the symptoms of the latter may predominate and mask those due to the pleurisy. The pain of pleurisy is as a rule local; occasionally, however, probably as the result of the irritation of the trunk of an intercostal nerve, the pain is referred to the abdomen This is most likely to occur in children, and may lead to a mistaken diagnosis of acute abdominal disease.

Kingston Fowler cites cases in which the referred pain has been felt on the side of the abdomen opposite to that of the pleurisy.

The pain varies very much in severity, at times being very acute, at times scarcely noticeable. It may be very severe when the diaphragmatic pleura is involved; in this condition the pain is usually referred to the line of the tenth rib and along the insertion of the diaphragm; hiccough, vomiting and dysphagia may accompany this form of pleurisy.

Apical pleurisy, which is so commonly associated with pulmonary tuberculosis even in its early stages, may cause no symptoms other than a slight aching in the shoulder, but this is of importance since it may lead to the discovery of disease in a stage at which treatment is likely to be successful.

Semeiology.—The signs of acute dry pleurisy vary somewhat according to the situation of the inflammation. In the common type of axillary pleurisy it is generally found that the movement of the affected side of the thorax is diminished; friction may be palpable, but as a rule no abnormal vibration is felt; vocal fremitus is unchanged unless there is a considerable exudate of lymph. Sometimes there is marked superficial tenderness of the skin; usually it is easy to demonstrate by firm pressure or percussion that there is deep tenderness over the inflamed area of pleura. The percussion note may lose some of its resonance and occasionally acquires a high-pitched "boxy" quality; this is to be explained by the defect in movement of the affected side and consequent incomplete expansion of the lung. On auscultation the breath-sounds are less well heard than usual and may be bronchial in quality; in typical cases they are associated with a to-and-fro rough friction-sound. This friction-sound is very variable; sometimes, especially in children, it is not heard, at other times a slight crackle is heard at the end of inspiration, which then abruptly ceases, the breath being "caught" for a moment before the commencement of the expiratory movement. This "catch" in the breath is apparently the result of a reflex inhibition of respiration when the pleural surfaces begin to rub together. In other cases the friction may produce crackling *râles*, which may be audible only in inspiration or both in inspiration and expiration.

Apical pleurisy is difficult to detect; we assume that it is present if there is defective movement, tenderness on percussion, and *râles* at the end of inspiration at one apex, but of necessity we are in doubt whether these are the signs of the pleurisy, or the underlying lung disease with which it is almost invariably associated.

Diaphragmatic pleurisy demands special consideration. The symptom of pain referred to the costal margin, as I have already said, is usually very acute. The patient prefers to sit in bed, leaning forward and resting his arms so that the accessory muscles may aid the costal respiration; the expression is usually anxious and the distress extreme. The respiratory movements are purely of the costal type, shallow and hurried, and limited on the affected side; often there is acatch at the end of inspiration; the diaphragm does not move in respiration, but the epigastrium is not sucked in during inspiration as it is when that muscle is

paralysed. Friction-sounds are seldom audible with the stethoscope. Associated with the thoracic signs there are usually some abdominal signs: the abdomen on one or both sides may be rigid and tender.

If, as is so often the case, the pleurisy is associated with fever, vomiting, hiccough and constipation, the simulation of acute abdominal disease may be very close, and it may not be until some days after the onset that the signs of pneumonia or of pleural effusion develop and establish the diagnosis.

Diagnosis.—It is not often that difficulty arises in the diagnosis of dry pleurisy; there are, however, certain conditions which require special consideration. Sometimes doubt may arise as to the co-existence of some associated pulmonary disease such as pneumonia.

In both pneumonia and pleurisy the onset may be sudden, with a chill or rigor, pain in the side and cough; in pneumonia, as a rule, the onset is more abrupt and the rigor more definite; in pleurisy the symptoms may take two or three days to develop and there may be a succession of slight chills. In pneumonia the constitutional disturbance tends to be greater and the temperature higher than in pleurisy; delirium is more frequent and the patient seems to be much more ill. Contrasting the victim of pneumonia with a sufferer from pleurisy it is found that the former presents more frequently the following signs: a hot, burning skin, labial herpes, a full, bounding pulse, of which the frequency is not increased in proportion to the respiration-rate, and a frequent cough with scanty, sticky, rusty sputum. In both conditions the urine is concentrated, but in pneumonia there is a marked deficiency in chlorides and albuminuria is common. The blood of pneumonic patients, if the resistance is good, has an excess of polymorphonuclear leucocytes; in simple dry pleurisy there is no characteristic change.

Of course it is only in those cases where the physical signs are equivocal that difficulty in diagnosis is likely to arise; it is well to remember when examining the chest that pneumonia usually has a lobar distribution and that pleurisy is not so limited.

Pathological conditions of the lungs associated with pleurisy may from time to time offer difficulties in diagnosis from simple pleurisy; experience teaches the wisdom of refusing to accept simple pleurisy as a final diagnosis until the ætiological factor has been determined. Local diseases of the thoracic wall may at times be mistaken for pleurisy.

Pleurodynia is a term used for a somewhat indefinite painful affection of the thoracic muscles, especially the intercostals, pectorals, and serrati; the pathology of the condition is obscure. Sometimes it seems to be due to the unwonted use of muscles out of training; sometimes there seems to be a fibrositis allied to the condition known commonly as muscular rheumatism; sometimes muscle-fibres have been ruptured by a sudden strain.

Whatever may be the cause of the pleurodynia, we find on examination a spot of localised tenderness over the muscle or its insertion; movements of the muscle are painful and there is a corresponding restraint in respiration. There is no constitutional disturbance, and of course the physical signs of pleurisy are absent.

Intercostal neuralgia has a most varied ætiology; it may result from implication of the spinal nerve-roots in diseases of the meninges as in spinal caries, syphilis and new growths, or there may be changes in the posterior root ganglia as in herpes zoster, or the trunk of the intercostal nerve may be attacked by neuritis or implicated in some disease of the chest-wall. Pain due to any of these conditions may simulate that of pleurisy. Pain in the side may also result from a functional disturbance; the referred pain of visceral disease falls into this group. It is usually associated with marked superficial tenderness without deep tenderness and is not necessarily aggravated by respiratory movements.

Actual disease of the chest-wall, such as tuberculosis or syphilis of the ribs or carcinoma of the breast, should not often give trouble in diagnosis; careful examination should lead to their immediate recognition.

Trichinosis of the intercostal muscles is usually mentioned in the list of conditions likely to be confused with pleurisy. I have not met with a case, nor do I think it likely under the present conditions obtaining in England.

Prognosis.—The prognosis of pleurisy depends almost entirely upon its ætiology. An attack of simple dry pleurisy usually results in early recovery unless it is associated with some serious underlying

disease. Occasionally it is followed by an effusion, serous, hæmorrhagic or purulent. These conditions will be considered later.

The remote prognosis of pleurisy in practice depends upon the nature of the infecting organism. If not tuberculous the prognosis is generally quite good. There may be some persistent adhesions which prevent free expansion of the lung on the affected side and so cause some dyspnœa and pain on exertion, and sometimes the chest-wall may fall in and spinal curvature may develop. Such deformity may render the patient more liable to bronchitis and other pulmonary diseases. It is said that occasionally the pleurisy may become chronic and lead to thickening of the pleura and to a spreading fibrosis of the supporting framework of the lung, this fibrosis leading to bronchiectasis. Cases proving such sequence of events are hard to find. If the pleurisy is tuberculous the prognosis is different. In practice we have to assume for safety that all pleurisy for which we cannot find an obvious cause is tuberculous. Treated reasonably there is no doubt that the majority of such cases recover and remain permanently well, but a study of the personal histories of consumptive patients shows that pleurisy is often the first evidence of tuberculisation, and that pulmonary infection ensues. Not infrequently cases occur in which it seems extremely probable that the pleurisy was inadequately treated, and we can but surmise that the subsequent pulmonary trouble might have been avoided.

I believe strongly that if there is any suspicion that a case of pleurisy may be tuberculous that it ought to be treated as long and as thoroughly and on the same lines as a case of early phthisis.

Treatment.—The treatment of dry pleurisy in the acute stage is largely symptomatic. Obviously an endeavour must be made to reduce the inflammation, but the only means at our disposal is the checking of mechanical disturbance of the inflamed parts. The direct application of cold in the form of an icebag or Leiter's tubes to the chest is said by some to check inflammation, but to me this seems extremely doubtful. Cold may act as an anodyne and relieve the pain, but it is difficult to believe that it can have any direct action on the inflamed pleura. We do not treat a superficial boil with an icebag.

A patient with acute pleurisy should be kept at rest in bed, and if the inflamed area is at all extensive it is wise to limit the movements of the affected side of the chest by means of strips of strapping passing obliquely from sternum to spine and spine to sternum, crossing in the axilla. In this way Nature's reflex limitation of movement may be effectually aided. Sometimes this strapping is not agreeable to the patient. If the hand, applied firmly to the chest, affords relief, strapping will be useful. It is obvious that strapping will be of little value if the chest is of an emphysematous type, the respiration being almost purely diaphragmatic. The cough in pleurisy is generally ineffectual, and it is harmful in that it disturbs the inflamed parts and causes pain. It should be checked by means of a linctus, which should contain opium if necessary. If the chest has not been strapped the pain may be relieved by painting with iodine, poulticing or leeching. These remedies are named in the order of increasing efficiency. Fever in pleurisy is seldom high and requires no special treatment. The patient will be more comfortable if the skin is moist, and sleep is very desirable. Dover's powder is an extremely valuable preparation at the onset of an attack of pleurisy.

Simple treatment on these lines is as a rule all that is needed unless an effusion or other complication develops.

The duration of the confinement to bed often requires the exercise of judgment. There can be but little doubt that rest is required so long as there is fever, and it is usually wise to enforce it if there is any pain. When the temperature has regained the normal level and the symptoms have disappeared, a gradual resumption of physical exercise may be permitted. In the majority of cases when the temperature falls and the symptoms disappear, the signs also fade away. This, however, is not always the case. Sometimes there may be persistent impairment of movement and dulness over the affected part. In these cases crepitus may remain audible for months, or even years, over the site of the pleurisy.

In such cases relaxation of strict routine must be gradual, and careful watch must be kept for the return of any symptoms which may suggest that harm has resulted.

Unless tuberculosis can be definitely excluded prolonged after-care with medical supervision is most desirable.

In the acute stage treatment by tuberculin is inadvisable, but in the later stages immunisation against tuberculin may be of advantage to the patient, and, if carefully supervised, is practically devoid of risk.

October 7th, 1912.

THREE DEMONSTRATIONS ON METHODS OF INVESTIGATING GASTRIC DISEASES.

Delivered at St. Bartholomew's Hospital.

By W. LANGDON BROWN, M.D., F.R.C.P.,
Medical Registrar to the Hospital, and Physician to the Metropolitan Hospital.

I. The Applied Physiology of Digestion.

Gentlemen,—It is obviously impossible, in the short time at our disposal, to go in detail into the whole question of gastric diseases, so I shall devote my attention more particularly, first to those points which we have learnt in more recent years from the application of some physiological methods to the study of digestive disorders, and secondly to those points which I have personal experience of.

I want to remind you that the anatomical conception of the position of the stomach has rather changed. The observations in the recumbent posture, which is what is always dealt with at operations and in the dissecting room where the stomach is unhardened, gave us an erroneous impression. From the hardened specimen of the stomach, which agrees well with the X-ray picture, we learn that the œsophagus enters a little below the highest point, the part above the œsophageal opening being always occupied by a certain amount of gas. Then comes the body of the stomach, mainly vertical, and separated from the pyloric portion by the incisura angularis. The pyloric part, as you see, comes a little below the umbilicus. That is another point which one was not prepared for. Corresponding with those anatomical differences, there is a marked difference of histology in the glands in the two parts of the organ. I exclude from this description the glands above the œsophageal opening, which are really only ordinary mucous glands and do not seem to play any active part in the digestive processes. The body of the stomach is occupied by highly differentiated glands, and these apparently are the only ones which secrete gastric juice. They are narrow-mouthed, and the ordinary lining of the stomach only goes in a little way into them, below which we meet with entirely different cells, the so-called chief cell, which is granular and secretes pepsin, and outside that the oxyntic cell, which is non-granular and secretes the hydrochloric acid. These glands either do not branch at all, or occasionally very slightly. They are simple glands. The pyloric glands are different; they are wide-mouthed and retain the same appearance as the lining of the stomach, except in the position of the nucleus, which becomes pushed up against the base of the cell. And these glands branch very considerably. Corresponding to this difference in structure, there is a marked difference of function. I have already said that these fundus glands are apparently the only ones which secrete pepsin and hydrochloric acid. The pyloric glands seem mainly absorptive, and, as we shall see presently, they play a very important part in the continuation of the gastric secretion after absorption has begun.

Gastric secretion.—Let us consider the nervous factors in gastric secretion. Our modern knowledge of this dates from the time and work of Pavlov, who divided the œsophagus in dogs, bringing the divided ends to the surface, and then separated off a part of gastric mucosa which opened separately on to the surface of the body. He found that whenever secretion was occurring in the large stomach it was occurring also in the small stomach, and he could collect specimens of gastric juice in that way which were uncontaminated by food. The right vagus was divided below the diaphragm at the time of the operation on the stomach—of course the right vagus does not supply the stomach to any large extent, but mainly goes to the intestines—while the left vagus which supplied both the large and the small stomach, was drawn into the wound in the neck, so that he could divide it easily when required. The result of this experiment was that when the dog took food into its mouth, although the food never got into the stomach at all, active secretion of gastric juice followed—the so-called "appetite juice." Clearly, then, there must be some nervous agency at work to produce it. And he proved that the nervous agency was this left vagus, because if he divided it, the effect of "sham feeding," as he called it, was completely lost: no secretion followed. On the other hand, gentle stimulation of the peripheral end of the vagus caused some secretion. If he adopted the method of direct feeding, putting food so that it went into the stomach without having gone through the mouth, sometimes there was secretion, and at other

times there was not, this depending apparently on the chemical nature of the food. We can arrange the articles of food into four groups according as they cause secretion or do not. I am giving you the results obtained by Pavlov, and subsequently by observers in the case of human beings after the operation of gastrostomy. Our first group consists of those which cause marked secretion. The greatest effect is produced by meat extract, apparently in consequence of the creatin which it contains. Next come condiments and spices, which cause marked secretion: I refer to such things as mustard, pepper, cloves, curry. Dilute alcohol caused a distinct secretion of gastric juice, and so did fluids containing CO_2. In the second group are those which cause slight secretion—milk and gelatin. According to Craven Moore, tea also causes some secretion. Thirdly, there are those which cause no secretion, and in that group must be placed the greater number of our ordinary foods, such things as peptones, white of egg, starch and sugar, if free from extractive substances. Lastly, there are substances which definitely inhibit secretion. These are fat and sodium bicarbonate. The mechanism by which these chemical reactions are produced was shown here some years ago by Dr. Edkins. He found that meat extract, if mixed with an extract of pyloric glands and injected into the circulation of a fasting animal, produced a secretion of gastric juice, while extract of the fundus glands did not have this effect. He also divided the stomach into two by means of a tampon and showed that the secretion occurred only at the fundus end, and not at the other. Evidently, then, a chemical stimulant to gastric secretion is produced by the action of this first group on the pyloric cells, and the chemical substance formed, sometimes spoken of as gastric secretin, is carried by the circulation to the fundus glands, and stimulates them to renewed activity. And if you come to think of it there is a necessity for some secondary mechanism of this sort. The gastric secretion is initiated by the taste of the food in the mouth, and also by appetite, because if the dogs even saw the food only, without being allowed to eat it, they still had some secretion. But when the appetite is satisfied and the food is no longer tasted, some mechanism is required to continue the secretion, and this is provided by the absorption of some of the food by the pyloric glands.

Certain practical deductions follow. The old saying that "hunger is the best sauce" is very well illustrated by these experiments. With two dogs Pavlov put 100 grms. of meat into the cul-de-sac he had made from the stomach. In the case of one dog nothing else was done, and in the case of the other the dog was allowed to keep up active sham feeding through his mouth. At the end of an hour the meat was withdrawn, and the amount which had been digested was estimated. It was found that in the first one, where nothing else has been done, only 6 grm. of the 100 had been digested in the first hour; whereas in the second one, that is to say where sham feeding had been kept up at the same time, 30 grm. of the 100 had been digested. In other words, the rate of digestion in the hour was increased five-fold by letting the dog taste the food. This secretion of "appetite juice" is an important fact, which explains what we know so well in practice, that you may work out a dietary on approved scientific principles for a patient, but may find it does not agree. The fact is one has to study the idiosyncrasy, the likes and dislikes of one's patient, and not attempt to force a diet upon them simply on theoretical grounds, because in that case they are deprived of the secretion of their "appetite juice." These experiments further throw some interesting light on the use of meat extracts. The late Sir Andrew Clark was accustomed to forbid all his patients soups, on the ground, he said, that these soups diluted the gastric juice. But as a matter of fact the gastric juice is hardly present at this stage, and one could quite well believe that the warm fluid containing little to digest, but with a stimulant to gastric juice in it, was quite a good thing to start a meal with. But his advice was often followed by satisfactory results, and I think that was due to the fact that many of his patients were busy professional men who suffered from excess of hydrochloric acid; soup increased the secretion of acid, of which they had already too much. We can also explain by this the discrepancy between the nutritive value of meat extracts and their apparently successful action in some cases. Their nutritional worth, so far from being accurately represented by the posters on the hoardings, has been estimated, I think by Dr. Robert Hutchison, to be equal to a teaspoonful of milk in a glass of water. But what they do is to stimulate the secretion of

gastric juice. We have to remember that in meat extracts we are not giving food, but preparing the way for the digestion of the food which should follow. But that function makes them useful in certain cases, as we shall see.

Another point is the action of the inhibitory substances. Fat is generally regarded as indigestible, and we now know that it positively inhibits gastric secretion. Why parents are so fond of insisting on their children eating fat I have never been able to understand: they always loathe it, and they can get their required carbon in a more pleasant form as carbohydrates, which they like. It may be good discipline, but it is bad physiology to force them to eat fat. We can also see how the chef has often anticipated the discoveries of the physiologist. In the civilised state, meals are often taken without much appetite, and in excessive amount, and the chef has learnt that he can artificially stimulate the appetite by hors d'œuvres and soups, which cause the secretion of gastric juice. There is also rationale for the taking of carbohydrates at the end of a meal in the form of sweets. For then the stomach contents may become different in reaction at the two ends, the first portion of food having been pressed over to the pylorus where it undergoes peptic digestion, while the food last consumed is retained in the fundus and remains alkaline for some time, so that salivary digestion can continue. A final important deduction from these results is to be found in the different effect of bitters clinically and pharmacologically. The pharmacologist puts bitters into the stomach and finding them cause no secretion of gastric juice tells us that they do no good. But as practical men we know that they do appear to benefit the patient. The point is that the bitter acts not on the stomach, but on the mouth. It is the stimulating effect of it on the mouth which causes the secretion of gastric juice. There is one difficulty which these experiments present, and that is with regard to sodium bicarbonate. For years we have been giving sodium bicarbonate before meals for the relief of dyspepsia, and we think with good results. But we are told now that so far from stimulating the secretion of gastric juice, sodium bicarbonate positively inhibits it. The explanation seems to be this: that the person who is benefited by alkalies before meals has considerable excess of mucus in the stomach, a catarrhal gastritis, and alkalies being solvents of mucus clear it off the mucous membrane before the food is taken, enabling digestion to begin, as it were, with a clean slate.

Movements of the stomach.—This is a subject on which we have acquired a very large amount of information in recent years through the agency of the X rays, and it surprises me to find that an eminent authority has put forward that X rays seem to him unnecessary and unpractical in the treatment of diseases of the stomach. I hope to show you by some skiagrams that very practical information can be thus obtained. I am accustomed to give large quantities of bismuth for the purpose. The amount of bismuth which is given for the purpose of taking X-ray photographs has been steadily increased up to four to six ounces of the carbonate or the oxy-chloride of bismuth. These never produce toxic effects, although the subnitrate may. It is given, according to the purposes of the examination, either with ground rice or in gruel or milk. It is of course necessary, before examining a patient in this way, that he should not have bismuth as a medicine for two or three days beforehand, otherwise the outline of the shadow will be obscured. For some of these skiagrams I am indebted to Dr. Jordan, formerly in charge of the Electrical Department at the Metropolitan Hospital, and for the others to Dr. Finzi, who now holds that post. Let me show you first the extraordinary difference between the appearance of the stomach with the X rays and the position which the stomach seems to adopt when you use any method of percussion or auscultatory percussion. An observation made by Dr. Hertz, of Guy's Hospital, illustrates this point. Three different observers mapped out what they believed to be the position of the stomach by means of auscultatory percussion, and their observations agreed. A bent wire was placed on the patient to indicate the position which they made out the stomach to occupy. The patient was then given bismuth. You now see the great difference in the position as shown by the X rays and that ascribed to it by the physicians who practised auscultatory percussion. It is of course important that in all cases the stomach should be examined with the patient both in the standing and in the lying-down posture. The omission of this precaution in the early days led to some very erroneous conclusions. In the recum-

bent posture the stomach tends to adopt an hour-glass shape, simply from the weight of the bismuth on either side of the vertebral column. When we give bismuth to the normal individual in the erect posture we find that the bismuth passes without any peristalsis at all, simply by the effect of gravity, through the fundus. And after a few minutes, peristaltic waves begin at the junction of the body of the stomach with the pylorus. The waves gather strength as they go on, and after a few minutes you see one wave pass through the pylorus, and the shadow begins to appear in the duodenum.

Now let us take some departures from these normal appearances and show what definite changes occur in the shadow in certain diseases. In each of these the position of the umbilicus is marked by a piece of metal or by a penny, so that you can see where the shadow lies in reference to it. The characteristic feature of malignant disease in this X-ray picture is that the shadow is broken up; the outline is a very irregular one. We can also see clearly the changes in gastroptosis. Compare with the normal stomach this one, which is a dropped stomach without dilatation. The vertical diameter is increased, while the horizontal is diminished. The greater curvature reaches down to the pubes. On the patient lying down, the fluid all gravitates up into the upper part of the stomach. In atonic dilatation of the stomach we have, instead of that appearance, a great flattening out of the shadow, an increase in the horizontal diameter producing a broad meniscus, and the peristaltic waves are feeble or altogether lacking. Now let me show you some skiagrams from examples of hour-glass stomach, which X-ray photographs have proved to be much more common than we had any idea of. I will first take some cases of genuine hour-glass stomach which were confirmed by operation. Notice a narrowing of the neck between the two halves of the stomach with a pouch above the constriction which is a characteristic feature. Next we have a spasmodic contraction producing the appearance of an hour-glass, which passed off under an anæsthetic. This is generally evidence of a gastric ulcer in the neighbourhood; it is a protective spasm produced by the irritation of the ulcer. In this next skiagram, however, the spasm was apparently secondary to old appendicitis. There was no evidence of ulcer in the case, though ulcer is the commonest cause of this spasmodic condition. There is another condition which is closely allied to hour-glass stomach. If a patient gets an ulcer on the lesser curvature of the stomach in the pyloric half it is liable to contract adhesions to the liver. The stomach is then tied up at that spot and hangs down on either side of it. But this disappears largely when the patient takes up the recumbent position, whereas the real hour-glass stomachs do not alter much with change in the posture of the patient. Here is a skiagram of a case in which the stomach was held up in that way, and the condition was confirmed by operation. I will show you the picture of another case which, I think, illustrates the value of the X-rays in diagnosis. A gentleman, æt. 60 years, was sent to me for anæmia and wasting, and I detected a lump in the epigastrium. The question arose whether it was in the stomach or where it was. Almost certainly it was of malignant character. The symptoms pointed to the stomach, but the X-ray picture proved it could not be there or in the transverse colon, and therefore it must be in the thin layer of the left lobe of the liver overlying the stomach, and that was where it proved to be. He was given a meal of bismuth, and the outline of the lump was marked out with a wire. You see that the shadow of the stomach is not interfered with in any way where that wire lies. Twenty-four hours later he was examined again, by which time the bismuth was in the transverse colon. Knowing now that the lesion was not in the stomach, I gave him a Seidlitz powder in two halves, so to as distend the stomach. You can now see the clear area produced by the distended stomach, and you can see the shadow of the transverse colon sweeping down in the region of the umbilicus; it is obvious that the wire which marks the position of the lump is not connected with either of these. So we were able to prove that the lump was not in either of these organs.

Types of dyspepsia.—I now want to pass to the recognition of certain types of dyspepsia, and their connection with alteration of the gastric secretion. The amount of hydrochloric acid is responsible to a very large extent for the kind of symptoms which are present. Let us consider what are the functions of the acid of the gastric juice. In the first place, pepsin is inactive in a neutral medium: the hydrochloric acid is essential to the activity of

the pepsin. Secondly, hydrochloric acid is an antiseptic, and, therefore, in its absence fermentative changes are much more likely to occur. Thirdly, it hydrolyses starch to some extent, as does any other mineral acid. Fourthly, it regulates the opening of the pyloric sphincter; and fifthly, it is the main stimulant to pancreatic secretion. So that if there is an absence of hydrochloric acid in the stomach there cannot be digestion, while fermentation will go on rapidly in the absence of that antiseptic. What might we expect if there were excess of hydrochloric acid? First we might expect that there would be some pain, but I am bound to say we cannot correlate the degree of pain with the degree of excess of hydrochloric acid. But the pain comes on when the stomach is getting empty, and, therefore, if the patient gives the history that pain comes on sooner after a light meal and at a longer interval after a heavy one, we may be fairly certain that the patient has an excess of hydrochloric acid. There may be pain within an hour of afternoon tea, while the patient may feel perfectly well after evening dinner until he is awakened by pain in the small hours of the morning. Then the pain is so severe that it is sometimes thought to be due to angina pectoris. There is probably, associated with this pain, some degree of pyloric spasm, because when the highly acid contents get into the duodenum they keep the pylorus closed. Thirdly, there may be pyrosis, which is a symptom of great significance in the diagnosis of hyperchlorhydria. I want to keep the meaning of that term strictly within the limits given to it by Sir William Roberts. It is often spoken of as if it were the same thing as regurgitation from the stomach. It often goes with it, but strictly speaking, pyrosis is the gush of alkaline saliva which occurs into the mouth during these attacks of pain. The significance of it is this: it appears to be the protective measure: it is Nature's attempt to neutralise the excessive acidity of the contents, and, therefore, it is only met with in cases of hyperchlorhydria. Like all pathological attempts at repair, it overshoots the mark, and the saliva is poured out in such quantities that it cannot be swallowed and so must be ejected. But a little inquiry will enable us to distinguish easily between the gushes of alkaline saliva which have a definite significance, and the water-brash which may occur in fermentative conditions as well. And the last point which is characteristic of cases of increased hydrochloric acid secretion is the appetite for indigestible things. You will often find the patient deliberately selects those articles of diet which you naturally suppose would be most indigestible. I remember some years ago, before I recognised the significance of this, lunching with a man who had complained all the morning of indigestion. He ate a large quantity of fat with his meat, butter with his potatoes, he had fruit tart with cream, and finished up with a large quantity of rich cheese. I said, "If you do not have indigestion after that, you deserve to." But he replied that he felt very much better after such a meal, and he was so for some hours. The fatty food inhibited his gastric secretion for a time, or at any rate diminished it, and he was more comfortable. But digestion has to be accomplished later; the pain is only delayed and the trouble returns. Having noted the kind of symptoms which you may expect according as the secretion is diminished or increased, let us consider what are the diseases in which these respective changes occur. Of course the gastric secretion is inhibited in acute gastritis, but that is always referable to the ingestion of something which should not have been taken. Diminished secretion or achlorhydria occurs in chronic gastritis in which it is simply the HCl that is reduced. In mucous gastritis there is + mucus and — HCl, and in the rare condition of atrophic gastritis, or as it is more often called achylia gastrica, associated with fibrosis of the stomach walls, both acid and pepsin will be absent. The other important condition in which this occurs is malignant disease. The hydrochloric acid may also be considerably reduced in many other conditions of extreme cachexia. Now as to the conditions in which increase of hydrochloric acid is found, that is to say hyperchlorhydria. First, there does seem to be a condition of simple hyperchlorhydria in otherwise healthy people; or what is sometimes called sthenic dyspepsia. Of late it has been claimed, particularly by Moynihan, that all these cases of hyperchlorhydria depend on an organic lesion, and that that lesion is generally an ulcer. I do not think that we can go as far as that, but I do feel that you ought not to make the diagnosis of simple hyperchlorhydria until you have excluded the other conditions in which hyperchlorhydria occurs. So we must consider what these other diseases are. First there is peptic ulcer, either in the stomach or in the duodenum. Next, and this is a point which has been overlooked until recently, hyperchlorhydria may result from conditions lower down in the alimentary canal; and I believe largely because they excite pyloric spasm. If an intestine is wounded experimentally there is inhibition of movements for some distance above and if the injury is high enough up there is also spasm of the pylorus for several hours. In gall-stones, appendicitis, and colitis, we often find the quantity of hydrochloric acid is increased. My suggestion is that these

diseases act in this way by causing a sort of protective spasm higher up, which is usually at the pylorus, and therefore prevents the escape of hydrochloric acid from the stomach, but is sometimes in the middle of the organ as I showed in that case of spasmodic hour-glass stomach.

We can often get an idea as to which of these two types of dyspepsia a patient is suffering from by careful consideration of the symptoms. You will find the contrast between the types vigorously drawn by Dr. Leonard Williams in his 'Minor Maladies.' Sufferers from achlorhydria are usually weakly, or convalescing from illness. And these people are often found to be excessive tea drinkers. The hyperchlorhydrics are usually strong, active and energetic, unless the condition is associated with an organic lesion. They are seldom teetotalers. Then with regard to the mental state, in achlorhydria the characteristic thing is depression and lassitude, whereas in the hyperchlorhydric type there is more probably irritability. The appetite is capricious, and generally poor with achlorhydria, but it is voracious with hyperchlorhydria, especially for indigestible things. The patient may not gratify that voracious appetite, but you will find on inquiry that it is there. With regard to the tongue, I ought to be able to make a very definite statement, but its appearance is variable. However, I may say that a brownish fur on the back of the tongue is nearly always associated with diminished hydrochloric acid. Sometimes there is glazing of the front of the tongue, whereas in the other form, that is to say hyperchlorhydria, there is usually a silvery fur in the front. But I feel some reluctance in making this statement because it is one of the things which I have not found to be constant. Then with regard to the character of the pain; in achlorhydria there is always more or less discomfort, increased immediately after food, whereas in hyperchlorhydria pain comes on as the stomach is emptying, the so-called hunger pain. Of course there may always be things which complicate the clinical picture. For instance, if there is an ulcer in the stomach the pain will very likely come on soon after food, and it is commonest in weakly anæmic girls, although there is hyperchlorhydria. Complications of that sort interfere with the clinical picture.

There must always be cases, however, which cannot be diagnosed by such simple considerations, where recourse must be had to more elaborate methods such as the test meal and the X rays. In the next demonstration I shall deal with the test meal and the methods of obtaining and examining gastric contents. I shall lay most emphasis on the simplest tests which I believe to be reliable, and hope to show how we can obtain light in doubtful cases by these chemical considerations.

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A LECTURE ON FLAT FOOT.

Delivered at St. Bartholomew's Hospital.

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Anatomical.

THE bones of the foot form four arches. Two of these are, however, completely filled in by soft parts, so that there is no arch apparent in the complete foot, although the chief weight is borne upon the extremities. The arches are best described as the internal and external longitudinal, the internal horizontal and the transverse.

The internal longitudinal arch is the most important, it is formed by the os calcis, astragalus, scaphoid, internal cuneiform and first metatarsal bones, its extremities form the chief points of support of the foot. Its summit is at the astragalo-scaphoid joint. The external longitudinal arch is formed by the os calcis, cuboid and fifth metatarsal with its summit at the calcaneo-cuboid joint; it is firmer than the internal and is filled in by the soft parts, so that it yields a continuous impression in a foot-print. It seldom gives way.

The transverse arch is formed by the metatarsals, it also is filled in by soft parts. The internal horizontal arch is formed by the inner border of the foot, so that when the feet are placed in contact, the heels and the heads of the first metatarsals touch, but the intermediate part of the feet show an intervening gap. In the strong foot this gap is still present in the standing position, but in weak feet it may disappear or the foot may even arch in the opposite direction. So that the region of the heads of the astragalus touch before the other parts of the inner border.

The internal longitudinal arch is that which is liable to obliteration, with it the horizontal arch is also invariably affected. But the latter may disappear or be reversed in cases in which the longitudinal arch is intact. Such cases, although they closely resemble flat foot, are more properly classed as instances of valgus deformity.

The internal arches are supported by ligaments and muscles. The principal ligaments concerned are:

The inferior and external calcaneo-scaphoid

ligaments which form a sling beneath the head of the astragalus, the long and short calcaneo-cuboid, and the plantar fascia.

The transverse arch is supported by the transverse metatarsal ligament.

But these ligaments would not long succeed in sustaining the weight to which the arches of the foot are subjected if it were not for the support which is given by the muscles. The action of the chief of these is as follows :

The tibialis anticus attached to the internal cuneiform and base of the first metatarsal bones lifts the longitudinal arch directly upward.

The tibialis posticus wrapping around beneath the summit of the internal longitudinal arch takes a great part in sustaining it, the tibial muscles are also responsible for maintaining the horizontal arch.

The flexor longus hallucis, the other long flexor tendons and to some extent the short muscle of the sole all assist in pulling backward the anterior pillar of the longitudinal arch, and so act the part of ties in sustaining this arch.

The gastrocnemius and soleus have no direct action upon the arches, but indirectly they are of great importance. If the tendo Achillis is unduly short so that the heels just fail to reach the ground, a severe strain is put upon the longitudinal arch. For by everting the foot the distance to the attachment of this tendon is shortened, and the heel is enabled to reach the ground, but in this position of the foot the tibial muscles are put upon the stretch, and if the position is maintained a severe degree of flat foot is almost certain to arise.

The peronei have an important action upon the arches. The peroneus longus passing as it does across the sole to its insertion, holds up the transverse arch. But in addition both this muscle and the peroneus brevis abduct the fore part of the foot, and in so doing depress the longitudinal arch. This action of the peronei is well seen in cases in which they are paralysed, for then an excess of the longitudinal arch is always seen, together with shortening of the whole foot and inversion of its fore part.

Movements.

The gross movements of the foot commonly occurring are extension and flexion of the ankle, and inversion with adduction of the foot (varus) and eversion with abduction (valgus).

As anatomists have confused the terms extension and flexion as applied to movements of the foot, it is necessary to define these terms. By extension in the correct (comparative) anatomical sense is meant dorsal extension, carried out by contraction of the tibialis anticus, long extensors and peroneus tertius muscles. In this movement the angle which the foot makes with the leg is lessened until at the limit of movement it is 60°. The opposite movement plantar flexion is carried out by the gastrocnemius and soleus assisted by the long flexors, tibialis posticus, and peronei. This movement is possible until the angle between the leg and foot is 150°, so that the whole range of movement is 90°.

The ankle-joint in these movements is a pure hinge-joint allowing no lateral movement whatever. There is, however, one small but important peculiarity. The lateral articular facet on the astragalus for the internal maleolus is longer and is carried further forward than is the corresponding facet for the external malleolus. In full dorsal extension of the foot the internal maleolus is carried right forward to the full extent of its facet, the external malleolus is, however, held back by tension on the posterior fasciculus of the external lateral ligament. As a result the longitudinal axis of the astragalus is altered in full dorsal extension, the head of the bone pointing more inward. The effect of this rotatory movement is to produce involuntarily a slight in-toeing in full dorsal extension ; a movement that is seen in walking when the leg is fully bent over the foot, just before the heel leaves the ground.

The movements of varus and valgus are very complex. The ankle-joint takes no part in either of them, but commonly the varus movement is combined with plantar flexion at the ankle, and valgus movement with dorsal extension. In the movement of varus the foot is adducted, the fore part is depressed and carried inward, the sole faces inward and the longitudinal arch is increased. It is carried out by the tibialis anticus and posticus muscles, assisted by the long and short flexors of the toes.

In the movement of valgus the foot is abducted, the fore part is raised and carried outward, the sole faces downward and slightly outward, and the longitudinal arch is depressed. It is, however, quite impossible to abolish this arch by any

voluntary or forced movement in the normal foot. These movements of varus and valgus as already stated are composite ones, practically all the tarsal joints being concerned; the chief part of the movement, however, takes place at the sub-astragaloid and mid-tarsal joints. At the mid-tarsal joint in addition a certain amount of plantar flexion of the fore part of the foot can take place, thus enabling the dorsal surface of the foot to be brought into a straight line with the front of the leg.

Attitudes.

In defects of the arch of the foot as in other static deformities, habitual attitudes are an important causative factor. In standing upon the inner border of the foot the peroneal muscles and extensors are contracted, the tibials are stretched and an extra strain is thrown upon the internal longitudinal arch, which tends thus to be depressed. The horizontal arch is usually obliterated or reversed. In standing upon the outer border of the foot the tibial and long flexor muscles are contracted, the peronei and extensors are stretched, and the internal longitudinal and horizontal arches are braced up. In general, when the feet are kept parallel or the toes turned in, the weight is thrown upon the outer border of the foot; when the feet are everted, on the contrary, the weight is thrown upon the inner border. Hence the strong position of the foot is that in which the toes are inverted.

In walking or running also the inverted position is the strong position of the foot, for the tibial muscles are kept in action and the arch well braced up.

The regulation of standing and walking attitudes, and the encouragement of the in-toeing position, is an important element in the treatment of patients suffering from weak or flat feet.

Flat Foot.

Flat foot is not a simple entity, capable of treatment by rule of thumb. The two elements, the mechanical and pathological, which enter into the causation of all deformities, must be borne in mind in every case, and their relative importance estimated. Thus it is evident that treatment which is appropriate for a young man of spare build, lacking in muscular tone, may be entirely unsuitable for a middle-aged stout woman; and flat foot resulting from an infective arthritis of the tarsal joints, or from paralysis of the tibial muscles naturally cannot be treated by the same means as flat foot due to purely mechanical causes. Ignoring, however, for the moment these factors, and looking at the problem of treatment from a purely mechanical standpoint, it is convenient to divide cases of flat foot into three classes according to their severity; further, it is most convenient to deal with the third or most severe degree first.

Rigid or Third Degree Flat Foot.

In the most severe cases of flat foot movement at the tarsal joints is greatly restricted or even completely abolished. This rigidity is in the first instance due to spasm of the peroneal and extensor muscles, although later peri-articular adhesions may still further restrict mobility, and in cases of very long standing there may even be changes in the shape of the tarsal bones which permanently affect movements. In a flat foot of a third degree then the internal longitudinal and horizontal arches are abolished, the astragalus may make a prominence upon the inner border of the foot, so that the tread may be widest opposite the head of this bone. There is often much pain, which may be diffuse or may be felt chiefly up the outer side of the foot and ankle. The walk is very stiff and guarded, and the patient is quite unable to move his tarsal joints, to invert his foot or to bring back any sort of arch. Inversion of the foot is not even possible when the foot is manipulated by the surgeon, and in fact when the spasm is severe, attempts at inversion are extremely painful.

The first step in treatment is evidently to get rid of the muscular spasm and to restore the mobility of the foot. In the slighter cases a few days rest in bed will often suffice to do this, in the more severe cases it is necessary to abolish the spasm by anæsthesia, and to fix the foot in the inverted position in plaster-of-Paris. In cases in which there are adhesions forcible wrenching of the foot under an anæsthetic may be necessary before the proper shape can be restored, fixation in plaster for from a fortnight to six weeks is then necessary. In a few cases there is a great tendency to recurrence of the spasm, and tenotomy of the peronei and extensors is advisable. The proper shape of the foot being restored, massage must be carried out during a further period of rest, until free passive movements of the tarsal joints are obtained. The

foot has now been reduced to the next stage of the deformity.

Second Degree Flat Foot.

In the second degree of flat foot movements are not restricted. Inversion of the foot and reconstruction of the arch can be carried out by the hands of the surgeon, but the patient cannot, when standing raise the arch by his own muscular effort. This test is best carried out by making the patient stand upon tip-toe. In the normal foot tip-toeing at once raises the arch to its full. A patient with flat foot of the second degree may be able to raise himself upon tip-toe, but he will do so without altering the shape of his foot.

At this stage the indication is to assist and strengthen the muscles which support the arch, but these muscles are not yet fit for strengthening by active exercises. The methods to be adopted are massage of the leg, active resisted movements to exercise the tibial and flexor muscles, and support to relieve the strain upon these muscles. Support may take two forms: it may be simply some device designed to throw the foot on to its outer border, and so relieve the tibial muscles, or it may consist in a pad or plate placed beneath the arch to support it directly.

In many cases, particularly in young subjects, a time arrives when the patient is able again to stand upon tip-toe with a proper arch raised by his own muscles. The next degree of the deformity is then reached.

First Degree Flat Foot.

In the first degree of flat foot the arch gives way when the foot is passively bearing the weight of the body, but the patient by an active contraction of the tibial muscles can reproduce a good arch. This is a stage at which active exercises to strengthen these muscles are necessary. To give active exercises in the standing position before this stage has been attained is mere waste of time, for the patient is quite incapable of carrying them out. It may also be necessary to provide a support for a flat foot of the first degree, but this will depend upon special circumstances, such as the age or weight of the patient, his work, etc.

Methods.

Some of the methods of treatment already alluded to require further mention. Inversion of the foot in plaster must be carried out under full ether anæsthesia, which must be maintained until the plaster case has hardened. The foot should first be thoroughly wrenched until all movements are free, and especially until the arch can be fully reconstructed with the foot at a right angle to the leg. If there is any difficulty on account of shortening of the tendo Achillis this tendon must be lengthened subcutaneously. The foot is then fully inverted, placed at a right angle to the leg and the plaster applied from the toes to well up the calf. The foot should be kept thus for at least a fortnight; if it has been necessary to lengthen the tendo Achillis or to divide the peronei, the plaster must be kept on for six weeks.

Several methods may be used to throw the foot on to the outer border. The simplest is to apply a clump (an extra thickness of leather) to the inner side of the sole immediateiy behind the tread of the ball of the great toe, this may be combined with a similar thickening of the fore and inner part of the heel. Or the heel may also be carried forward under the instep. But this is not necessary if the bootmaker strengthens the waist of the boot suffiicently. If a stronger support is necessary a steel spring may be fixed in the outer side of the boot (concealed valgus spring). Or to get greater strength still a short outside iron with a valgus T strap may be fixed to the boot.

Plates and pads for direct support of the arch are made and sold in great variety. They should give slightly to pressure, be not too high, and fixed correctly so as to raise the arch in the proper place. They are often fixed too far forward. For simply supporting the arch an india-rubber pad is as good as anything. Some forms of plate serve the double purpose of supporting the arch and throwing the foot onto the outer side at the same time. The best of these is the Whitman brace. This is a steel plate which should be specially made for each patient upon a plaster cast of his foot, and fitted into a boot made large enough to take it. It has the defect of being cumbersome and difficult to make well, but in some cases of flat foot in middle-aged patients it gives great and permanent relief.

Exercises for second degree flat foot should be combined with massage of the leg-muscles and are best given by a trained masseuse, inasmuch as they consist in properly designed resisted movements.

Exercises for the first degree may, however, be taught and carried out at home. Simple and useful ones are :

(1) Stand with heels together, toes separated by an angle of 60°. (1) Rise on tip-toe ; (2) separate heels, keeping toes firmly fixed on the ground ; (3) bring heels together ; (4) lower heels.

(2) Stand as before. (1) Rise on tip-toe ; (2) keeping heels raised, bend knees sinking slowly until the position of squatting on the heels is almost reached ; (3) rise again slowly, straightening knees ; (4) lower heels.

(3) Stand as before. Rise on tip-toe. Walk forward and backward slowly on tip-toe.

(4) Stand as before. Tip slowly over on to the outer side of the feet as far as possible. Maintain this position for a short time and then slowly return.

All these are best carried out whilst wearing soft heelless slippers.

Choice of Method.

Before deciding on the line of treatment to be adopted, every case of flat foot requires careful consideration. In the first place an accurate diagnosis must be made. It is safe to say that at least a third of the patients commonly said to suffer from flat feet have no such condition. To make sure of the condition of the arch, a print of the tread should always be taken, this is very easily and quickly done by making the patient tread first upon a pad of felt soaked with a solution of potassium ferrocyanide, and then immediately upon a piece of blotting paper which has been soaked in ferric chloride solution and dried. A prussian blue print of the tread is thus obtained. Many feet apparently flat will be found to give prints which are practically normal in appearance, these are cases of valgus deformity, that is of eversion and abduction of the fore part of the foot at the sub-astragaloid and mid-tarsal joints, without any dropping of the arch. Valgus may exist in the simple (first) or in the rigid (third) degree, it requires practically the same treatment as the corresponding stage of flat foot, but the prognosis as regards recovery is better, and direct support to the arch is not necessary. It is not uncommon to find that a supposed flat foot proves when a print has been taken to be overarched. Overarched feet may give at least as much trouble as flat feet.

The next step is to exclude an arthritis of the tarsal joints. What is apparently a simple flat foot may prove to be symptomatic of a tuberculous or gonorrhœal arthritis of one of those joints, and an error of diagnosis may lead to a catastrophe. Then any local cause of pain on pressure upon some part of the foot, or on movement must be looked for. Two of the commonest of these are hallux rigidus and a painful corn at the base of the fifth toe. Paralysis or paresis of any of the muscles and contraction of the tendo Achillis must also be looked for.

If the case is of the third degree, treatment may now be commenced forthwith, the methods already described for restoring the arch and regaining mobility being used. As soon as the second degree has been reached, however, the age, weight, social condition and work of patient must be taken into account. In a young patient who need not work, treatment may be successfully carried out by massage and exercises without the provision of any support, or with simple clumping of the boot. In older persons support is more likely to be required particularly if they are of heavy build. Whilst in those who have to follow a laborious occupation to attempt to treat a case of flat foot which has been of severe degree without a support is to court failure. In no condition is a careful consideration of the individual needs of the patient more necessary than in flat foot. And indeed it is frequently only by trial that it is possible to secure the greatest amount of comfort for the patient.

October 9th, 1912.

West London Post-Graduate College.

Mr. Donald Armour, Vice-Dean of the West London Post-graduate College, informs us that the Winter Session of the College commences on October 14th. The opening address will be given on Monday, October 14th, at 5 p.m., by Dr. Seymour Taylor, Consulting Physician to the Hospital, on "Medical Education — Then and Now." The West London Hospital in the Hammersmith Road is of easy access by rail, tube, and motor omnibus.

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ON SYPHILIS OF THE NERVOUS SYSTEM IN INFANCY, CHILDHOOD, AND EARLY ADULT LIFE.

By SYDNEY A. OWEN, M.D., M.R.C.P.,

Assistant Physician to the West London Hospital and Queen's Hospital for Children; and Physician to Out-patients, City of London Hospital, Victoria Park.

THE *Spirochæta pallida* is the infective agent of syphilis. This organism can be found frequently in the primary and secondary lesions of the acquired disease, less constantly in its tertiary lesions. Further, it can be demonstrated by suitable methods in nearly all the tissues, more especially the liver and spleen, of the still-born fœtus, or of the child who dies shortly after birth of the so-called "inherited" or "congenital" malady. Syphilis can be communicated to suitable animals by the injection of material obtained from active, especially primary, lesions, or by the injection of semen which contains the spirochæte long after the primary sore has healed (Levy-Bing, Finger, Landsteiner).

Even an infant may be the subject of the acquired disease. This is by no means exceptional: numerous cases have been reported. For the most part an infected wet-nurse or even the child's own mother, who has become infected subsequent to the birth of the child, is responsible. In such cases the course of the disease differs in no way from that of the inherited type (Mott).

In childhood, too, extra-genital chancres acquired in a variety of ways are by no means exceptional (Leiner).

In by far the greater number of cases the child contracts the malady indirectly, being born of parents one or both of whom are suffering from syphilis. In such cases we have to deal with the

so-called "congenital," " "inherited," or "hereditary" type of the disease. Of these three terms "inherited" is permissible only in the sense that something is received from the parent. Since the discovery of the *Spirochæta pallida* by Schaudinn and Hoffmann in 1905, our views as to the transmission of syphilis from parent to child have, of necessity, undergone modification. It can no longer be regarded as hereditary in the true scientific meaning of that word (Pernet). The subject is, in fact, a very vexed one (Marshall). I am inclined to the view that, if not invariably, at least in the majority of instances, the infant *in utero* is directly infected, *viâ* the placenta, from the mother, who is, or has been at one time, the subject of acquired syphilis. In mild infections or where the virus has been attenuated by time, the placenta appears to act as a barrier to the passage of the spirochæte, in whatever stage of its development, or to its toxin. Under these conditions the mother, though the subject of syphilis herself, begets a normal child. Where the infection is recent or severe this is not so: an abortion, a macerated fœtus, a premature delivery, or a syphilitic infant results. To my mind the word "congenital" would be appropriate were it not for the fact we associate with this word conditions, symptoms, and signs which are manifest at birth. In not a few instances the signs of inherited syphilis manifest themselves for the first time months, or it may be even years, after birth. Up to that time the child is in good health so far as one can determine. Syphilis hereditaria tarda may be met with even after forty years of age (Poulard).

Colles's law.—It is a well-established fact that a syphilitic infant may be born of a woman who has shown no active signs of syphilis during her pregnancy. This infected child, although it will give a healthy wet-nurse syphilis, will not infect its own mother; the mother is immune. This is known as Colles's law. Well-authenticated exceptions to this law have been brought forward from time to time. On the other hand, many eminent authorities maintain the law is absolute.

As already stated, the parental transmission of syphilis is a very vexed subject, but by many well-known persons it is held that whilst transmission in the majority of cases is through the father, less frequently it is "maternal," and in a few cases it is "mixed." When the infection is "paternal" the organism gains access presumably in the semen to the uterus, and directly invades the ovum or the tissues of the developing fœtus. The mother may be infected simultaneously, manifesting primary sore and secondaries in the usual way. On the other hand, when the infection is paternal only the mother appears to escape. At the same time it is supposed certain hypothetical substances pass to her from the infected child, because although she bears an infected child that infant will not give her syphilis.

At the present day, however, we have a means of showing that the mother of such a child is suffering from syphilis despite the fact she never presented any signs of active disease. The Wassermann reaction of the blood in these parents is positive (Knoepfelmacher, Bauer, Dean). It is only right to state that the interpretation which I put upon this latter fact is not accepted as such by everyone (Still, Marshall).

Profeta's law.—Let me remind you of another interesting law. It is said that if a healthy woman becomes infected during a normal pregnancy her child, though it presents no sign of the disease, is itself immune. This is Profeta's law. I have no doubt in my own mind the blood-serum of such a child would give a positive Wassermann reaction. The disease exists, but is latent.

We are more familiar with the oft-quoted statement, that it is very exceptional for a person the subject of the inherited disease to fall a prey to acquired syphilis. There are several fallacies here. It is quite possible that the apparent rarity of such cases is to be partially explained by the fact few adults show conspicuous traces of the "inherited" disease (Hutchinson). Transmission to the third or fourth generation is difficult to prove or disprove. It is denied by most who have the more intimate knowledge of the facts. Cases certainly are published from time to time which appear to admit of the possibility (Boeck, Suggett).

There are several other interesting problems connected with the transmission of this disease I should like to touch upon, but it would be digressing too far from my subject proper. There is one, however, I should like to lay stress upon. It is this: Whereas we are all familiar with certain types of disease of the nervous system occurring in young subjects who present undoubted evidence of the inherited or acquired disease, I am con-

vinced another important group exists where the lesions are in reality due to syphilis, although the child presents none of the classical signs which usually bear witness to this. The main object of my lecture is to attempt to prove and to emphasise this very important fact. It must be that the toxin of syphilis modifies the spermatozoon or the ovum, or both, in such a way that the embryo resulting from their union is profoundly modified from the normal during the course of its development. The nervous structures of such an individual especially are apt to show evidence of arrested development, early decay or even maldevelopment. If this is the case syphilis is capable, in the "inherited" malady at any rate, of producing dystrophies, abiotrophies and lesions comparable to those seen in other diseases of the nervous system which we believe are not due to syphilis. Some of the lesions of tabes and general paralysis may be of this nature. Developmental anomalies of the brain in juvenile general paralysis have been described (Trapet).

Syphilis and infant mortality.—It behoves us as medical men to pay great attention to the adequate diagnosis and treatment of syphilis in all its forms. The acquired disease cripples many individuals mentally and physically. The offspring of syphilitic parents suffer still more. The disease is responsible for a considerable amount of sterility among women otherwise healthy. It has been calculated that, on the average, in 46 per cent. cases pregnancy ends prematurely in syphilitic women. The infant mortality is 42 per cent. To quote actual figures: Charpentier in 781 cases of syphilis found the pregnancy interrupted in 302 (42 per cent.). Fournier in 200 pregnancies in 100 syphilitic women found abortion or premature labour in 140 (70 per cent.), the average frequency of abortion (all causes) being approximately one to five pregnancies. The date at which syphilis is contracted by the mother, relative to the date of conception, is very important. Of 90 women who had become pregnant during the first year after infection, 50 aborted or had children born dead, 38 gave birth to children who soon died, and 2 gave birth to children who survived. Such figures as these must convince us that the potential as well as the actual loss of useful lives in any given period must be very considerable.

The incidence of lesions of the nervous system in the inherited disease.—Of those children who give positive evidence of syphilis, about 10 per cent. (Still) and 13 per cent. (Rumpf.) manifest at one time or another signs of some lesion of the nervous system. The probability, then, that the nervous system will be involved is by no means low, although all are agreed that the central nervous system stands low on the list of the order of frequency in which the various organs are involved in the child who is the subject of the inherited disease.

Moreover, although the nervous structures are infrequently attacked, we must never lose sight of the fact that when involved the lesions are multiple, extensive, and more often than not severe.

The diagnosis of syphilis in the infant and young subjects, and the determination as to whether the lesions of the nervous system are due to syphilis.—In practice there are two important questions we may be faced with: (1) Has this child syphilis? (2) Are the nervous lesions in a syphilitic child due to syphilis?

Any child, the subject of the acquired or the inherited disease, may be found to be suffering from a lesion of the nervous system which is certainly not due to syphilis. I have under my care at the present moment an infant who presents unmistakable signs of the inherited malady. The child also has a typical Erb's palsy of the upper arm type. The presentation was a breach one and the labour a difficult one in consequence. Delivery of the after-coming head with traction on the arm will satisfactorily account for the lesion. Suitable treatment has given a satisfactory result. On the other hand there are certain lesions of the nervous system which, whether they occur in the adult or a child, are certainly due to syphilis or its late so-called meta-syphilitic effects. I refer to tabes and general paralysis. An individual who suffers from one of these diseases need give no confirmatory history or signs of syphilis. We now know they must have had syphilis. In fact we expect none. It is a well-known fact that persons suffering with tabes and general paralysis rarely manifest tertiary lesions or other evidence of syphilis on the body. The co-existence of specific lesions with tabes and general paralysis has, of course, been noted by several observers (Mott, Rivaillon and others). When we are dealing with lesions which may be due to other poisons it is difficult sometimes to determine whether syphilis is responsible

or not. If it were possible to demonstrate with certainty during life the spirochæte in the tissues of the nervous system the problem would be much simplified. This is manifestly impossible, and unfortunately, too, the cerebro-spinal fluid rarely contains the spirochæte (Dupérié, Neisser, etc.), or if it does, a means to demonstrate it has yet to be discovered.

In young subjects who are suffering from inherited lues we may attempt to arrive at a diagnosis and the determination of the two questions above along the following lines:

(*a*) A careful inquiry into the *family history* may be of value. All are agreed that too much stress must not be placed upon abortions, miscarriages and premature births. An impartial review of the family histories of the cases narrated in this paper shows that only one, so far as abortions are concerned, gives evidence of positive value. I am forced to conclude, too, from a consideration of these family histories that it is by no means exceptional for healthy children to "slip in," so to speak, between the diseased ones. A careful search among the other members of the family may reveal another child who presents undoubted signs of the inherited disease. In a small percentage of cases the parent presents evidence of the acquired malady. On the other hand the parent may be a general paralytic or a tabetic. A negative history in the parent is of no value. Moreover, where the child is suspected of syphilitic taint and gives a positive Wassermann reaction (blood-serum) the mother's serum, although her previous history would appear absolutely to exclude infection, may also give a positive reaction. On looking through the literature I find Stiefler has placed on record a remarkable instance of familial and juvenile tabes following acquired syphilis.

(*b*) The evidence of *stigmata*, some of which are concealed only because they need to be looked for. The importance of a thorough ophthalmoscopic examination cannot be too frequently insisted upon. It may reveal primary atrophy, choroidal, vitreous, or retinal changes which afford valuable evidence of syphilis. Changes in the deeper layers of the cornea should be looked for, as they give sometimes evidence of previous interstitial keratitis. The Argyll-Robertson pupil of the classical type is strong evidence of syphilis, and more especially of parasyphilis (Babinski, Mott, Michell Clarke, Henderson and others). On the other hand too great stress must not be placed upon such signs as cranio-tabes or enlarged liver and spleen. Sclerosis of the testicles, deafness without suppuration, enlarged epitrochlear glands (Reiche), Moon's molars, blue sclerotics (Rolleston), are some features that may assist in the diagnosis.

(*c*) If, however, we wish to obtain conclusive evidence we must turn to a Wassermann reaction of the blood and a detailed examination of the cerebro-spinal fluid.

(1) *The Wassermann reaction of the blood.*—From the standpoint of practical medicine I believe this reaction when positive to be of real value. I agree with those who state a negative reaction is inconclusive. There are other diseases, mainly protozoal, which give a positive reaction, *e.g.* trypanosomiasis, piroplasmosis, ? palludism (Levaditi), noma (Butler), frambœsia or yaws, but the diagnosis of these conditions in this country at any rate should not present any difficulty. The reaction must be performed by a competent person according to one, or other, of the well-recognised techniques. The test in my cases has been carried out by my colleague, Dr. Woodforde, at the Queen's Hospital. The method employed is the one originally introduced by Wassermann.

(2) *The cerebro-spinal fluid.*—(*a*) *Chemical.*—The normal fluid, I would remind you, "is a clear fluid with a specific gravity of 1·004 to 1·007. The reaction is alkaline, and it flows from the cannula at the rate of one drop per second. It is non-toxic and non-virulent. It contains a trace of globulin but no true albumen, lipoids or choline. Sugar exists to the extent of 0·15 to 0·18 per cent. Urea amounts to 0·15 per cent. It contains a small percentage of chlorides, carbonates and potassium salts" (Mott).

Under pathological conditions the more important differences to which I would draw your attention in this connection are: (1) The increase in the amount of globulin and the presence of albumen; (2) the presence of lipoids and choline in excess. This is very characteristic of certain degenerative diseases of the nervous system; (3) an excess of reducing substance is sometimes seen, more especially in tubercular meningitis; (4) increased tension.

(*b*) *Cytological examination.*—The normal fluid is almost free from cells of any kind. It is not

unusual to find 2 to 5 per c.mm. Counts above 5 are considered by some to denote pathological excess (Assmann).

Under pathological conditions a polynuclear or mononuclear leucocytosis occurs, and this is a feature of considerable importance from a diagnostic point of view. A polynuclear excess points to the invasion of the sub-arachnoid space by a pathogenic organism other than the tubercle bacillus. (Exceptionally, more especially in its early stages, even in tubercular infections a polynuclear count exists.) A mononucler excess points to syphilis, parasyphilis and tuberculosis. It is found also constantly in sleeping-sickness. Rarely it is found in the following conditions: mumps, herpes, leukæmia, chloroma and poliomyelitis (Mott). The presence of plasma-cells is especially associated with syphilis of the nervous system, and more especially the parasyphilitic diseases. Where hæmorrhages have taken place macrophages are occasionally met with.

(*c*) *Organisms.*—In pathological states these occur. Unfortunately for the purpose of the diagnosis of syphilis of the nervous system, the specific organism has rarely been found (Neisser, Dupérié, Sézary). It offers a marked contrast in this respect to sleeping-sickness. Here the organism can much more easily be detected.

(*d*) *The Wassermann reaction of the cerebro-spinal fluid.*—The application of the Wassermann test to the cerebro-spinal fluid may assist us considerably. Stated briefly a positive reaction points usually to a meta-syphilitic affection of the nervous system. A negative reaction is strong presumptive evidence against this type of lesion.

In any case, then, of syphilis, or rather suspected syphilis of the nervous system, a careful examination of the cerebro-spinal fluid is essential. It will not only tell us in a large percentage of cases whether we are dealing with a syphilitic infection, it will give us information as to the type of syphilitic infection present.

Briefly summarised, the more important points to bear in mind, having excluded tubercular meningitis and sleeping-sickness, are the following:

(1) A very high lymphocyte count (250 to 300 cells per c.mm.) points to an active gummatous meningitis which may subside on active treatment with mercurials. Dr. Mott, in his recent Oliver-Sharpey lectures, refers to a good instance of this type of case.

(2) A high count (50 to 150 cells per c.mm.) points to active syphilis or meta-syphilis, *e. g.* tabes or general paralysis. In these two diseases the increase in the lymphocytes is early, and occurs before definite physical signs enable one to establish a diagnosis beyond dispute.

(3) In pseudo-tabes, the well-known clinical picture resembling the true disease dependent upon a tertiary, meningo-myelitic, rather than a para-syphilitic lesion, although the count is high the result of treatment will diminish the number of leucocytes. In tabes it is doubtful if this is so.

(4) In the meta-syphilitic diseases the lymphocytosis precedes as a rule the positive Wassermann reaction in the cerebro-spinal fluid. Further, a positive reaction has been found apart from any increase in the lymphocytes. The positive character of the reaction cannot therefore be wholly, if at all, dependent on the presence of a lymphocytic excess.

(5) In active syphilis of the nervous system the Wassermann reaction of the blood-serum is positive, but negative in the cerebro-spinal fluid. This statement is not absolute. In acute cases of luetic infection both blood and cerebro-spinal fluid may give a positive reaction (Henderson). In both tabes and general paralysis the Wassermann reaction of the blood-serum is positive. The reaction in the cerebro-spinal fluid in general paralysis is nearly always positive, and in tabes it is positive in a very high percentage of cases (average 70 to 80 per cent.) (Smith, Candler, Mott, Muirhead, Assmann and many others).

(6) All forms of luetic infection of the nervous system cannot be excluded by the above means, because in a few cases the fluid may be normal. This does not apply, however, to the meta-syphilitic diseases (Assmann).

(7) Conditions which are not specific may be very misleading. Henderson reports the case of a male patient who died as the result of an endothelioma in the region of the hypophysis. The patient, æt. 40 years, gave a history of syphilis, and the Wassermann reaction of the blood and the cerebro-spinal fluid were both positive. In addition to this there was a lymphocytosis and an excess of globulin. At the post-mortem, however, there was no evidence of syphilis or meta-syphilis.

I wish to bring before you to-day the clinical records of twelve cases. The Wassermann reaction of the blood has been carried out in eight cases. The evidence, direct and indirect, of syphilis and the part it plays in these eight cases is as follows:

(1) All give a positive Wassermann reaction of the blood.

(2) No one gives a positive reaction of the cerebro-spinal fluid. This, as I hope to show later, is what I should expect. From it you will conclude among the eight cases I refer to I have no instance of tabes or general paralysis to bring before you.

(3) Three give a positive blood reaction, and presented at one time or another signs which are, as a rule, considered to be due to syphilis: *e.g.* Daisy A— (Case 10), typical Hutchinsonian incisors, Moon's molars, and unequal, irregular pupils, with sluggish reaction to light; Marjorie B— (Case 3), a positive blood reaction and typical condylomata; Harold C— (Case 1), specific hyalitis in addition to a positive blood reaction.

(4) One child, Susan J— (Case 9), gives a positive blood reaction, and the cerebro-spinal fluid shows a lymphocytic excess with the presence of albumen and globulin in excess. The pupillary reaction to light is sluggish, the family history is very suggestive, and there has been distinct improvement on mercury and iodides.

(5) In one case, Amelia D— (Case 6), the blood reaction was positive, the cerebro-spinal fluid gave a slight excess of lymphocytes and a small cloud of albumen. A von Pirquet's reaction was negative, and no tubercle bacilli were found in the fluid removed by lumbar puncture. Although there are no other signs to substantiate the diagnosis of the acquired or the inherited disease, the symptoms and signs rapidly cleared on the administration of mercury and iodides without the addition of any iron. Moreover, it would be difficult, I think, to find any other more satisfactory cause to account for the course of the illness in this child.

(6) In the case of Annie E— (Case 4), and Stanley F— (Case 5) and Ellen G— (Case 2), the positive blood reaction is the only direct evidence of syphilis, and it would be fair comment, I admit, to say that these cases are instances of slight spastic diplegia with mental deficiency, of mental deficiency and fits, and of mental deficiency respectively occurring in children who are the subjects of the inherited disease and in whom this luetic taint is only accidental.

Of the four other cases, three (Cases 7, 8, and 12) came under my notice prior to the date when lumbar puncture and Wassermann reactions were in vogue. In the case of the patient Harry H— (Case 11), I have had no opportunity to carry out a Wassermann reaction of the blood. During the last few months Dr. Woodforde has carried out for me a Wassermann reaction in 42 cases of various kinds. For the sake of convenience I have made a rough classification into positive, weakly positive and negative groups.

Number of cases and sex.	Diagnosis.
A. *Positive:*	
1–8. M. 3 and F. 5.	—The cases narrated in this paper.
9. F., æt. 9 years.	—Typical case of inherited syphilis; anæmia, sabre tibiæ, glands, blue sclerotics; small pleural effusion; much improvement on anti-syphilitic treatment; reaction strongly positive.
B. *Weakly positive:*	
10. F., æt. 11 years.	—Atrophy of the kidney with marked arterial lesions, microscopically; hypertrophied heart, high blood-pressure; intense albuminuric retinitis and terminal secondary infections of the pleura and peritoneum (post-mortem).
11. M., æt. 5 mths.	—Inherited syphilis, clinically, with mucous tubercles, slight craniotabes, very marasmic.
12. F., æt. 8 years.	—? Congenital pulmonary stenosis; hæmaturia.
C. *Negative:*	
13–18. Æt. 4½–10 years.	—*Grand mal* or *petit mal*, or both combined.
19. M., æt. 4 mths.	—Clinically, basal meningitis and marasmus; no opportunity to examine the cerebro-spinal fluid.

Number of cases and sex.	Diagnosis.
20. M., æt. 6 years.	—Clinically, a typical diplegia; difficult labour with instruments; microcephalic; marked spasticity; backward.
21. M., æt. 2¾ years.	—Clinically, diplegia; instrumental delivery; squint; backward.
22. F., æt. 3 years.	—Clinically, ? cerebral diplegia; premature child, one of twins; cannot walk or talk; intense anæmia; vision probably defective.
23. M., æt. 8 mths.	—Clinically, hydrocephalus, anæmia, pneumonia.
24. F., æt. 11 years.	—Clinically, cerebral monoplegia, with athetosis of the left arm; the limb is shorter than the right; disability dates from birth; the mother was very ill during the pregnancy.
25. M., æt. 3 years.	—Clinically, cerebral diplegia, spastic paraplegia; cannot sit up or talk; labour natural; no miscarriage; is the fourth child; parents healthy; fundus is normal.
26. F., æt. 3¼ years.	—Clinically, mental deficiency; cannot talk and cannot walk; Jewish child; the only child; fundus is normal.
27. F., æt. 7 mths.	—Typical case of progressive spinal atrophy of infants; the first child; Werding-Hoffmann type; post-mortem.
28. M., æt. 7 years.	—Typical post-papillitic atrophy with poor visual acuity; no cause determined; X-ray of skull negative; no other signs in nervous system; measles, æt. 2; no drug responsible as far as can be determined
29. M., æt. 5 years.	—Case of miliary tuberculosis; disseminate tubercle of the choroid; persistent palsy of the sixth cranial nerve; cerebro-spinal fluid normal; persistent high temperature for several weeks; post-mortem confirms diagnosis; no tumour, but massive tubercles in the right Sylvian fossa; general miliary tuberculosis.
30. M., æt. 3½ years.	—Mongol; a Jew; mental deficiency; the twelfth child.
31. M., æt. 5 years.	—Clinically is not a true cretin, but is very backward and has congenital ptosis.
32. M., æt. 3½ years.	—Mental deficiency; recurrent jaundice; reaction twice negative.
33. M., æt. 8 mths.	—Clinically, amaurosis; ? congenital syphilis; ? retinitis punctata albescens; cerebro-spinal fluid is normal (on two occasions), and the Wassermann test applied to it is also negative.
34. M., æt. 9 mths.	—Typical case of amaurotic dementia; a Jew; the second case in the family.
35. M., æt. 11 years.	—Clinically, progressive paraparesis with atrophy of the peronei and spasticity; ? diagnosis
36. M., æt 10. years.	—Unilateral œdema of the leg and no albumen.
37. M., æt. 5 mths.	—Clinically, marasmus; ? inherited syphilis.
38–40.	— Three cases of intermittent jaundice with enlarged liver and spleen.
41. M., æt. 9 years.	—Clinically, enlarged heart; high blood-pressure; no albumen in the urine, no casts; fundus normal.

Number of cases and sex.	Diagnosis.
42. F., æt. 1 $\frac{3}{12}$ mths.	—Recent head injury; ? slight weakness of external rectus; cerebro-spinal fluid normal; meningism, recovery.

Pathological.—I now wish to bring before you the clinical rather than the pathological side of the subject. I have only partial evidence from one case to bring to your notice. I hope to publish the full account of the morbid anatomy of this one fatal case (Case 3) at a later date. It is stated the lesions found in the nervous tissues of the child suffering from inherited lues differ in no way from those which occur in the acquired disease (Mott). They are associated for the most part with one, or more usually with a combination, of three different processes, the disease being histologically characterised by (*a*) a primary toxic degeneration, (*b*) a small-celled infiltration, and (*c*) a reactive proliferation of connective-tissue elements (Ernest Jones). It is important to remember that many of the symptoms and signs which patients suffering with syphilis of the nervous system present are not due to primary implication of nerve structures, but to the indirect effects occasioned by vascular changes, meningeal and gummatous lesions. Further irregularity and variability are the two leading features of the effect of the syphilitic poison on all structures. This applies especially to the nervous lesions. In the acquired disease cerebral and spinal lesions are frequently associated together. Spinal lesions are of frequent occurrence. So far as I can judge from the literature on the subject pure spinal manifestations, apart from the tabetic picture, are exceptional in the inherited type of the infection. On the other hand, there are certain lesions, such as a primary parenchymatous neuritis of the spinal (peripheral) nerves which are of infrequent occurrence in the acquired as well as the inherited disease (Bonnet and Laurent).

Under the microscope the picture seen resembles very closely that seen in allied protozoal diseases, notably, sleeping-sickness. The perivascular lymphatics and the meningeal spaces are crowded with lymphocytes and plasma-cells. Hyperplasia of the neuroglial cells is characteristic. The blood-vessels show endarteritis in variable degree. The brunt of the mischief falls upon the sub-endothelial layers of the intima. Sclerosis with diffuse or patchy thickening results. The lumen of the vessel is gradually obliterated. The media and adventitia are involved, but to a less extent. Diminished blood supply, thrombosis with softening results. The pia arachnoid and dura show considerable infiltration and thickening. The former changes occur especially at the base, the latter on the convexity of the brain. In general paralysis the atrophy of the cortical substance is very characteristic.

By appropriate methods the spirochæte has been demonstrated in the several tissues of the nervous system in those children, the subjects of the inherited disease, who die shortly after birth or who succumb to active meningeal and cerebral lesions.

The characteristic changes in the cerebro-spinal fluid have already been referred to in a preceeding paragraph. It is interesting to note that even in those cases which are undoubtedly specific the spirochæte has rarely been shown (Neisser, Gaucher, Merle, Dupérié).

Fairbanks, in a lengthy paper, "Cerebral Syphilis in Children," has collected from numerous authors the records, clinical and pathological, of a large number of cases of cerebral and spinal lues. Those who are interested in the subject should consult this paper. In an abbreviated form it contains much that is interesting from the pathological as well as the clinical side.

Weyl has studied the condition of the cerebrum in syphilitic infants who have died from various causes. Certain well-marked changes were discovered although the patients had not, in every case, manifested any nervous symptoms. He raises the question as to whether such changes are not a constant feature and may not have some bearing on the mental processes of those who survive.

The age at which nervous symptoms first manifest themselves in the subject of the inherited disease.—This varies within considerable limits:

(*a*) During the first few months of life many without doubt succumb to the effects of hydrocephalus and meningitis. Now that lumbar puncture is a routine method of examination in a doubtful nervous case our knowledge on these matters has been considerably increased. Convulsions are a fertile cause of death in infants, and possibly many

so-called idiopathic convulsions should be attributed to syphilis if our powers of accurate diagnosis were extended.

(*b*) In a small percentage the parent seeks advice soon after the child's birth because her suspicions are aroused that vision is defective. In children who are older and have arrived at the age when they should commence to sit up and take an interest in their surroundings and attempt to grasp objects, it may be noted that the child does none of these things. In other cases the limbs are unduly stiff, the child lies curled up, and owing to this unnatural stiffness the parent's fears are aroused. A high degree of spasticity may be present, and yet the parent will affirm the condition never attracted her attention until a few days, or even hours, prior to the time she sought advice.

(*c*) When the child arrives at the age when he or she should learn to walk and talk, disability on the part of the child to do this cannot fail to attract the attention of the most unobservant.

(*d*) Epileptic manifestation may cause the parent to seek advice at any age.

(*e*) During the school period the child is brought because vision is defective, the child is backward and cannot learn, or because there are occasional fits. Interstitial keratitis occurs at definite age-periods with remarkable frequency. In passing it is well to remember syphilis is not the only cause of interstitial keratitis in young subjects. Further, it may be due to acquired lues.

(*f*) At or about puberty the manifestations of the meta-syphilitic diseases are especially liable to commence. On the other hand, we meet with a few cases of adolescent general paralysis at a much later date, eighteen to twenty years being a not infrequent age. Of course in some of these cases we may be dealing with instances of meta-syphilis of acquired origin.

(*g*) When we are dealing with the acquired affection it is important to remember that the nervous system is involved early, by no means infrequently within the first eighteen months of the date of infection. Not a few cases have been published in which nervous signs manifested themselves within a few months, or even weeks, of the date of the primary infection. Inasmuch as children are in no way exempt from the acquired disease the onset of nervous symptoms may occur at any age. When certain signs and symptoms we typically associate with acquired lues in the adult occur in a child we should not hesitate to attribute them to syphilis because they occur in a young child. The case of Amelia D— affords a good instance of this.

Inasmuch as mild attacks of acquired syphilis are so liable to be overlooked, and in children for obvious reasons this is even still more likely to occur, and inasmuch as nervous lesions are specially liable to be present in those individuals where syphilis has been untreated, it is extremely important to keep it prominently before us as a possible ætiological factor in the young in the causation of diseases of the nervous system. When the meta-syphilitic diseases occur they do as a rule at a date more remote from the primary infection. Now that we are all familiar with the occurrence of tabes and general paralysis in their characteristic adult form in quite young subjects the age of the patient should be no bar to an early diagnosis.

As already stated, parasyphilitic affections of the nervous system occur as a rule at a date more remote from the primary infection than do those conditions associated with tertiary manifestations. An average period is nine to eleven years. In rare instances, however, the interval may be much shorter. Marchand and Petit record the case of a young woman who contracted syphilis at the age of nineteen. She was untreated, and two years later mental symptoms developed. Specific treatment had no effect. The post-mortem revealed typical lesions of general paralysis.

(*To be continued.*)

October 14th, 1912.

Early Pharyngeal Symptoms of Poliomyelitis.—M. Neustaedter calls attention to the nasopharyngeal symptoms which he believes are invariably present in this disease. Frequently one is told that the patient begins to sneeze, or makes attempts to sneeze, and the mother or nurse suggests that the patient is developing measles. Upon inspection, however, there is not a red, sore, and hyperæmic throat, but rather an anæmic, glistening, œdematous condition of the pharyngeal mucosa, with a serous frothy transudate, quite analogous to the œdematous swelling of the cord. This condition the author has found to persist in the majority of the cases, even several weeks after the onset of the paralysis. This sign, in his opinion, is pathognomonic of the disease.—*Med. Rec.*, vol. lxxxii, No. 13.

LECTURES
ON
DISEASES OF THE PLEURA.*

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Lecture II.

The signs of pleural effusion are often easy to recognise, but occasionally the diagnosis may present great difficulties.

The most important signs are the absolute dulness of the percussion-note over the fluid, the loss of tactile vocal fremitus over a corresponding area, and the displacement of the heart away from the affected side.

Great value must also be attached to exploration with an aspirating syringe and the examination with the X rays; it is, however, desirable to arrive at the diagnosis, if possible, before resorting to these means of investigation. So far as I know there is only one pathological condition other than pleural effusion which may be associated with the three signs of absolute dulness on percussion, loss of tactile vocal fremitus, and displacement of the heart to the opposite side; that condition is a massive growth in the lower part of one lung. The condition is rare, and when it occurs the diagnosis is usually easy because other signs are present. At times, however, the diagnosis is impossible without the aid of the aspirating needle. Difficulties arise when the presence of the fluid is not associated with all these three signs, or their investigation is rendered difficult by some abnormality. The dull percussion-note is due to the damping effect of fluid upon the sonorous vibrations which result when the healthy chest-wall is struck. Solid lung and thickening of the pleura may also give a dull note, though it is seldom so dull as when fluid is present; moreover, a careful plotting out of the upper border of the dulness may give a clue to the correct diagnosis; collections of fluid between the liver and the diaphragm, as in subphrenic abscess, or in the liver, as in tropical abscess, may produce marked dulness at one base.

It may be extremely difficult to detect differences in resonance on percussion if the chest be covered with a thick layer of fat, or is œdematous. On the other hand, in thin people and in children percussion may give a resonant sound owing to the vibration of the opposite side of the chest; this difficulty is best avoided by cultivating the sense of resistance to the pleximeter finger, and in some cases by direct percussion of the chest-wall with the tips of three or four fingers at once, or even with the flat of the hand.

The fremitus of the voice is conducted through the bones of the healthy chest-wall, and felt by the examiner's hand; if fluid be present in the pleural cavity the conduction is damped, and the fremitus is no longer felt. Difficulties arise if the timbre of the voice is such that even in normal circumstances fremitus is not palpable; such conditions occur in women and in children, and it is then impossible to investigate the sign. In children the fremitus of crying is usually palpable, and should this *contretemps* occur at the time of the examination soothing efforts may be combined with an attempt to investigate the conduction of fremitus. Again, when the chest is covered with a thick layer of fat or is œdematous the conduction of fremitus may be so much impaired that investigation of inequalities is difficult, if not impossible.

Pathological conditions other than pleural effusion are associated with diminution or loss of vocal fremitus; thickened pleura, new growths, pneumothorax, subphrenic abscess may be confused with pleural effusion if too much stress be laid upon this isolated sign.

The displacement of the heart away from the affected side is a sign which merits very careful consideration.

In health the lungs are continually on the stretch, and by virtue of their elasticity each exerts traction on the other; in the post-mortem room, when the thorax is opened the lungs are found to shrink away from the chest-wall, and only partly fill the thoracic cavity. If the pleural surfaces on one side only are separated the lung on the corresponding side will tend to collapse; moreover, it will exert less traction upon the opposite lung; consequently the mediastinum will move over to the opposite side even though there may be no positive intra-pleural pressure. It has been shown by experiment that in pleural effusions unless they are very large and associated with complete collapse of the lung the tension is usually negative as measured by a manometer. In serous pleural effusions the displacement of the heart is conditioned by the elasticity of the lungs, and is

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seldom so great as in pneumothorax or empyema when other factors become important. Should the lung have lost its elasticity, as in emphysema, a serous pleural effusion, even of considerable size, may not be associated with any cardiac displacement. The presence of emphysema, therefore, deprives this sign of pleural effusion of its importance. In most cases of pleural effusion, however, the position of the heart, or for practical convenience of the apical impulse, is of great importance not only in the original diagnosis, but also in watching the progress.

There are many other signs which are frequently to be found where there is a pleural effusion, but partly because they may be absent and partly because they may indicate conditions other than pleural effusion their importance is subsidiary.

On inspection it is sometimes noticed that the respiratory movement of the affected side of the chest is less than that of the sound side; the frequency of this sign in other conditions detracts from its value as a sign of pleural effusion.

Bulging of the intercostal spaces, so common in pneumothorax and large empyemata, is unusual when there is a serous effusion. Litten's sign is dependent on the fact that when there is a pleural effusion the movement of inspiration does not strip the diaphragm from the chest-wall as the lung descends into the costo-phrenic sinus. Consequently there is not the sucking in of the intercostal spaces in succession which should occur in health and produce the moving shadow when the patient is observed under oblique illumination. If there is an effusion the costo-phrenic sinus is full of fluid and the shadow is not seen. If the lung is emphysematous it occupies the greater part of the costo-phrenic sinus even during expiration, consequently there is no moving shadow during respiration. If there be pneumonic consolidation, pleural adhesions, paralysis of the diaphragm or immobilisation of the diaphragm from other causes the shadow will not be seen. These reasons combined with the difficulty often experienced in arranging the illumination correctly diminish the value of Litten's sign. If, however, the shadow is seen on one side of the thorax and not on the other it may aid the diagnosis.

Mensuration may show that the chest is asymmetrical when there is a pleural effusion; most commonly the affected side of the chest is larger than the other, but this is not constant, and occasionally the semi-circumference is actually less on the side on which there is an effusion. The difference in measurement depends upon the intra-pleural tension, and when the effusion is serous this may be markedly negative; in pneumothorax, large empyemata and hæmothorax the tension is usually positive, and the semi-circumference on the affected side is then the greater. If the effusion be known to be serous and measurement shows that the side of the chest involved is larger than the other, we may assume that it is probable that the lung has collapsed completely and the effusion is a large one.

Palpation, as I have already mentioned, reveals the loss of tactile vocal fremitus over a pleural effusion; careful investigation shows that this loss is co-extensive with the effusion and corresponds exactly with the absolute dulness found on percussion. The ulnar border of the hand is convenient for the detection of changes in fremitus. It seems to be particularly sensitive to this form of stimulation. It is not infrequently assumed that vocal fremitus in health is chiefly transmitted through the lung and that fluid prevents this transmission; that this explanation is not complete is shown by the fact that in pneumothorax the fremitus, though generally diminished, is not lost in all cases.

It is not infrequently found that there is a line of deep tenderness just above the level of the fluid; this corresponds with the line of separation of the visceral from the parietal pleura, and on auscultation friction-sounds are often heard in the same situation.

Further details may be elicited by careful percussion. Where the effusion is large and reaches as high as the third rib the upper border of the dulness is horizontal; with smaller effusions the upper border tends to be curved as pointed out by Damoiseau.

Damoiseau's curve is higher in the axilla than at either the anterior or posterior extremities. The position of this line alters but little on changing the posture of the patient. Any marked and rapid alteration suggests that air as well as fluid is present in the pleural cavity. In such condition the upper border of the dulness remains absolutely horizontal whatever the posture of the patient.

When there is a pleural effusion the respiratory movements affect but little the level of the dulness; if the lung resonance is found to encroach in deep inspiration upon some abnormal dulness at the

lower part of the chest we are led to suspect that the disease is below the diaphragm.

The reason for this is obvious: if there is an effusion the fluid is in the costo-phrenic sinus, the lung is partly collapsed and descends but little during inspiration; if the dulness be due to an abnormal condition below the diaphragm there may not be any impediment to the descent of the lung into the sinus during inspiration.

Grocco's triangle of paravertebral dulness on the side opposite to the pleural effusion seems to have but slight diagnostic value; the explanation seems to be that the fluid occupying the vertebro-mediastinal pleural sinus acts as a mute to the bodies of the vertebræ and prevents them from conducting the normal pulmonary resonance.

In effusions of medium or large size the partial collapse or relaxation of the lung produces a peculiar quality in the percussion note above the level of the fluid; this peculiar note is known as skodaic resonance, and it is found in conditions where the lung is less aërated than in health though not quite airless—for instance, in the early congestive stage of pneumonia. The sign does not seem to have any diagnostic value in the case of pleural effusion; the note may change if the patient's mouth is open or closed during inspiration and expiration, and on altering the patient's posture.

On auscultation over a pleural effusion the breath-sounds usually are heard very feebly or not at all; if heard there is much diminution in the vesicular murmur, and sometimes there is distinct bronchial or even tubular breathing. The weakening of the breath-sounds can be explained partly by the defective air-entry into the affected side, and partly by the bad sound-conducting qualities of a partially collapsed lung. Fluid conducts sound well, and so does a solid lung; if the lung be completely collapsed the laryngeal breath-sounds may be heard as tubular or bronchial breathing over an effusion. At times consolidation of the superficial layers of the lung from collapse or pneumonia explains the audible bronchial breathing.

In children the lung has not lost its elasticity and complete collapse is probable; bronchial or tubular breathing is more commonly heard over pleural effusions in children than in adults. Experience has not confirmed Baccelli's statement that the presence of tubular breathing suggests that the fluid is serous rather than purulent.

If the breath-sounds over a pleural effusion are not well heard or are absent the vocal resonance will be diminished; if they are bronchial or tubular the voice-sounds may acquire a bleating quality: this is termed "ægophony." Laennec noticed that ægophony was frequently heard just above the level of a pleural effusion, and concluded that the sign was pathognomonic of the condition; we know now that ægophony may be heard over lung-tissue which is partly deprived of air either by collapse or congestion, and that the sign does not necessarily indicate the presence of fluid in the pleural cavity.

The examination of the thorax by means of the X rays may be of great value in the diagnosis of pleural effusion. An efficient screening apparatus offers much help in difficult cases. A pleural effusion is opaque to the rays, but there seems to be no difference in opacity between serum and pus; the upper border of the shadow is as a rule not very sharply defined; it is usually concave, with the concavity upwards and inwards.

Subphrenic disease shows a shadow, of which the upper border is convex upwards; in hydro-pneumothorax the upper border of the shadow is always horizontal, and succussion waves can be demonstrated.

In pulmonary disease the shadow is usually more diffuse and has an outline quite different to that of a pleural effusion.

The position of the heart may be determined by the screen, and the information thus obtained may be of great value.

I show you now four cases which illustrate some of the points to which I have alluded.

The first case is in a boy, æt. 3 years, whose chest was aspirated on August 13th, but still contains a large quantity of fluid.

It is possible to demonstrate two of the three chief signs—dulness and displacement of the heart; fremitus we cannot investigate without making him cry. Of the subsidiary signs you will note that the right side of the chest, where the effusion is, looks larger and moves less well than the left side. Measurement shows that the half-girth on the affected side is $\frac{1}{2}$ in. more than on the sound side ($10\frac{1}{4}$ in. against $9\frac{3}{4}$ in.). Palpation shows that the liver is displaced slightly downwards. The percussion dulness extends to the third rib in front and the fifth rib in the scapular line behind; the upper border is not quite straight,

being slightly higher in the axilla, and remains almost in the same position whatever the posture of the child. The breath-sounds and voice-sounds are inaudible over the dull area.

The effusion in this case is serous and contains small lymphocytes.

CASE 2.—This boy is æt. 12 years. He was admitted to the hospital with a pleural effusion on the left side, and 24 oz. of serum have been removed.

Examination reveals the signs of dulness, loss of fremitus and displacement of the heart. The affected side of the chest looks smaller and moves less well than the sound side; the half-girth is ¾ in. less than that of the right side (13¼ in. to 14 in). Over the dull area neither breath-sounds nor vocal resonance can be heard.

CASE 3.—This girl, æt. 24 years, still has well-marked signs of pleural effusion, though 24 oz. of serum were removed the other day.

The left side of the chest is absolutely dull up to the second rib, and over the dull area vocal fremitus cannot be felt; the cardiac dulness extends well to the right of the sternum. There is no marked asymmetry of the chest, though mensuration shows that the affected side measures ½ in. less than the sound side (14¾ in. to 15¼ in.).

On auscultation bronchial breathing is heard over most of the left side, and the vocal resonance is increased.

From a consideration of the signs it seems probable that there is a large effusion, and that the lung is completely collapsed.

CASE 4.—The next case is that of a girl, æt. 15 years, who has a pleural effusion on the left side, of which 16 oz. were removed by aspiration the other day.

The signs of dulness, loss of fremitus and displacement of the heart are present.

The affected side of the chest is flat and moves poorly; measurement shows that the half-girth is 1¼ in. less than on the right side.

Weak bronchial breathing is audible over the dull area in the axillary region, and at the upper border of the dull area crepitus is audible with inspiration and expiration. The fluid is undergoing absorption, the collapsed lung is re-expanding, and the roughened pleural surfaces are again coming into contact.

October 14th, 1912.

A DEMONSTRATION OF GENERAL SURGICAL CASES AT ST. BARTHOLOMEW'S HOSPITAL.

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I DO not intend to go fully into any particular subject to-day, but to show you some of the cases at present under my care, making use of each as a text for some, possibly rather disjointed, remarks.

Here is a small boy who, until quite recently, was running about with a well-marked spinal curvature due to tuberculous disease. It is unusual for a boy to get to that stage and yet keep on his feet without complaint of pain. He should have been prevented from walking some time ago, but now we must make the best of the deformity, which should not have been allowed to occur. And the best that can be made of this is that the spine should become fixed by bony ankylosis in its faulty position, a result which will only be achieved by keeping the patient on his back for 18 months or more.

And here let me briefly comment generally on two questions which you are sure to be asked when you have decided to put a tuberculous joint at rest. How long must the part be kept fixed? What will be the functional result?

With regard to the first question the wisest plan is not to give an actual date, but to require a period of at least six months after the cessation of all pain. In this present case, as the patient has practicallly no symptoms, I have been obliged to put it vaguely as eighteen months or more.

With regard to the second, What will be the functional result? The answer must depend upon the stage the disease has reached. If the articular cartilages have been destroyed cure can only result by ankylosis of the exposed bony surfaces, and your care must be to see that this takes place in the best position.

In the present case this will perpetuate the deformity. For although some little time ago the proposal was made to straighten out deformed spines it came to nothing, as it necessarily must have done, being opposed to the two main principles that if the articular cartilages have been destroyed cure can only result from bony ankylosis, and bony

ankylosis can only result from the bones concerned being kept in apposition.

If, however, the disease has not progressed to the destruction of the articular cartilages the movements of the joint may be completely restored.

Between these two results of complete mobility and complete fixation you will often meet with cases of restricted movement which will tax your resources and make you wish you could act with the freedom from responsibility enjoyed by the bone-setter.

If you employ forcible movements you may achieve a brilliant success by breaking down a few old adhesions which interfered with perfect function, or you may court disaster by lighting up a local or possibly a general tuberculosis.

You must therefore exercise the caution which your responsibility demands, but caution need not necessarily mean inaction.

This next patient is a man who has had attacks of renal colic.

Several skiagrams have been taken of the renal region but no stone is shown.

On examination his urine is found to contain large quantities of oxalate crystals. You should bear in mind the fact that oxaluria will sometimes give rise to symptoms every bit as marked as those which may be caused by the presence of an actual stone, such as increased frequency of micturition, pains, and profuse hæmaturia.

Sometimes the appearance of the crystals may be directly connected with some article of diet. For instance I know a man who sometimes gets hæmaturia from this cause after partaking of strawberries.

This patient is an illustration of what not to do. He constantly passes stones. He has been in the hospital on many occasions, and has had stones removed from his right kidney, his bladder and urethra. A few days ago he came in with a stone impacted in his urethra just in front of the scrotum.

Now if there is a stone in the penile urethra it should be extracted through the meatus if possible, if not it may be pushed back into the membranous urethra and extracted through a perinæal incision or into the bladder and crushed, the latter being the better plan; it should not be removed by an incision in the penile urethra, as this will almost certainly result in the formation of a fistulous track.

You will see, however, that he has an incision in the ante-scrotal portion of his urethra from which, in spite of the fact that it was carefully stitched up with catgut, urine passes.

I was obliged to extract the stone in this situation as I could not get it out *viâ* the meatus either whole or in pieces, and it lay in a sort of pouch which stopped it from going back.

The next patient came to the hospital on account of a lump in his neck which he had noticed for three months and which was getting larger; it gave him no pain or trouble of any kind and it was the increase in size which led him to seek advice. On questioning him he admitted his throat had been a little sore for the past month or so, but he had not thought this worthy of complaint.

Examination at once revealed the fact that the lump in his neck was due to a hard fixed carcinomatous mass involving the submaxillary set of lymphatic glands, secondary to a small epithelioma of the fauces. This condition, where the secondary infiltration of the glands first attracts the patient's notice, is by no means uncommon in the neck, and the primary focus which passes unnoticed may be difficult to find, such as a small extrinsic growth in the larynx or an epithelioma of the œsophagus.

But I would not have you think that the absence of symptoms in connection with cancer is peculiar to these regions. I am accustomed to insist upon the fact that wherever it may arise early carcinoma has no symptoms.

The lurid pictures generally painted of the disease in which pain and loss of flesh and cachexia play an important part apply only to its later stages.

Your diagnosis must be made therefore on physical signs alone; there will be no symptoms to aid you—in fact, it is the absence of symptoms which will excite your suspicions and cause you to negative an inflammatory origin for the trouble in favour of new growth.

If only this fact could be widely spread among the public as well as the profession one would be spared the frequent spectacle of the patient who comes up with a neglected inoperable carcinoma, say, of the breast, for instance, with a happy smile on her face as she says, "I know it is nothing serious because I have had no pain."

One more point before we pass on: you must not wait for a secondary growth before you make a diagnosis of the nature of the primary. This may

sound an unnecessary caution, yet how often do we hear it said by people who ought to know better, "I don't think it's cancer, there are no glands"? Never feel for glands in connection with a growth which you suspect to be malignant until you have made up your mind about it. If you can get no further than a feeling of doubt the absence of palpable glands must not influence your proceedings: you must call the microscope to your aid at once.

The examination of the glands may, however, yield important results from the point of view of prognosis and treatment.

The next case is one of Pott's fracture.

This is a serious lesion because it is a fracture which is always complicated by a dislocation—a point one is apt to forget.

Now the modern treatment of fractures has been rendered more easy and at the same time more difficult by the introduction of operative measures and the use of the X rays.

With regard to the former I can only say in passing that no universal rule is applicable; some will be best treated by wiring or plating and some by the application of splints.

But in all cases of fracture and even of suspected fracture the employment of the X rays will always prove of the greatest value.

Now this patient will be treated in the way we commonly treat this variety of fracture in this ward. No attempt will be made to "set" the fracture until the patient is under an anæsthetic, then it will be manipulated into the best position possible and kept there with a back splint and two side splints. Then skiagrams will be taken, both lateral and antero-posterior views. If these show the position to be satisfactory, well and good; if not, the above may have to be repeated, and that more than once. "Counsels of perfection" you may say, "but impracticable in the ordinary round of practice."

Even so, give your patient the best advice at the time and leave him to declare its impossibility, rather than yourself assume it and run the risk, if in the end the result is not all that could be desired, of being blamed for having left anything undone.

Here is a man with a knee-joint which has been bad for six weeks. The trouble came on acutely, but now the acute symptoms have subsided the wasting of the muscles, the bulbous white swelling of the joint, the absence of fluid, and the thickening of synovial membrane and of the peri-articular tissues might well lead to a diagnosis of tuberculous disease of long standing. The amount of peri-articular thickening was almost as great at the end of a fortnight from the onset as now, and the early occurrence of this is perhaps one of the most characteristic features of this disease, which is that of gonorrhœal arthritis.

He has been treated with a sensitised gonococcal vaccine, receiving 20 millions on August 15th, 50 millions on the 16th, and 100 millions on the 17th. This produced a rapid improvement in the local condition, but as his articular cartilages have been eroded (shown by the occurrence of starting pains at night), bony ankylosis is inevitable.

This is a man who was admitted towards the end of the first twenty-four hours of an acute attack of appendicitis. We removed his appendix at once and he is now convalescent.

Now I am not one of those who advocate immediate operation in all cases of acute appendicitis at whatever stage of the complaint they may be first seen.

It is our ignorance of what is going on in the appendix which makes such a course advisable in the early hours, an ignorance which is shown by the inclusion of such widely different conditions as inflammation, perforation, gangrene, stricture and concretion under one common name—"appendicitis."

I hope and believe the time may come when we shall be able to discriminate between these conditions, and submit to operation only such cases as really require it, at a time which is always inconvenient and with an abdominal condition, which to say the least of it, is unfavourable for laparotomy.

I will not go further into this question now, but let me tell you of two conditions in which I believe immediate operation to be wrong.

If the patient is not seen until such time as a swelling has formed in the appendix region then the best plan of treatment is to wait.

The presence of a definite swelling is an indication that the mischief has become localised, and danger to life has passed; and although in the early stages a swollen, engorged, distended appendix may be easily removed with little disturbance to the surrounding parts and little bleeding, the case is quite different when it has become embedded

amongst thickened coils of intestine and covered over with recently formed vascular adhesions, which readily bleed at the slightest touch.

Again, when there are signs that a localised abscess has recently formed, wait; there is no object in opening it at once, but by waiting much may be gained.

Adhesions become more dense, and the abscess may adhere to the abdominal wall; but far beyond all other advantages is the fact that the organisms in the pus become less virulent, and in many of our cases they have been found to have died, so that cultures from the pus remained sterile. I have no figures at this moment that I can give you, but can state that by this expectant method of treating localised appendix abscesses we have reduced the death-rate from this cause to almost *nil*.

This man again illustrates what I have said just now about the absence of symptoms in cases of carcinoma. You will see he has an ulcerated growth in the front of the floor of his mouth under his tongue, which has infiltrated his lower jaw so that the teeth are loosened, and spread into his tongue, so that he cannot readily protrude it.

There is no salivation, no pain and no tenderness, so that you can all feel the hardness of the ulcer's margins without causing the patient to complain.

He states that the ulcer has been there a month, but from the advanced nature of the growth as well as the wide-spread infiltration of the glands on both sides of the neck, I feel sure that is an underestimate of its duration.

Be that as it may it is interesting to note though it seems almost incredible, that the condition caused him so little inconvenience that he sought advice for the first time a day or two ago when he came up here because his teeth were getting loose.

Now note the position of the ulcer and remember this, that any ulcer which starts on the under surface of the tongue along the fold of mucous membrane overlying the submaxillary duct is almost certainly a carcinoma. No other of the ulcers which affect the tongue, dyspeptic, dental, tuberculous, syphilitic, etc., start there.

The only one besides the epitheliomatous which has its origin at this spot is the ulcer which sometimes forms over a salivary calculus impacted in the submaxillary duct, and the hardness of the calculus may to the inexperienced suggest the hardness of malignant disease.

The next case shown was that of a young man who had dysentery a week after his return from India five months previously, and who was admitted to the hospital on August 20th with a liver abscess bulging in the epigastric region. Aspiration and injection of the cavity with a 10 per cent. solution of quinine hydrochloride, after exposure of the liver through a small incision, was followed by ½-gr. doses of emetine subcutaneously each day over a period of seven days, the dose on the fifth day being omitted, as recommended by Major Rogers, I.M.S. As a result of this treatment the leucocytes dropped from 28,000 on August 24th to 7000 on August 29th, the epigastric swelling disappeared, and the patient is now convalescent.

A woman upon whom the complete operation for removal of breast and glands had been performed five days previously showed the advantage of not tying the arm to the side, but putting it up in a position of full abduction, as she could now raise her arm so as to place her hand upon her occiput.

A woman, who had come up on account of a femoral hernia, and who was found to have in addition a large, apparently rapidly growing fibroid of the uterus, was shown to illustrate the fact that the increase in the size of the abdominal contents is an important factor in the causation of hernia, and the opportunity was taken to point out the value of a complete routine examination in all such cases before recommending a radical cure.

Several other cases were shown, including a woman upon whom appendicectomy had been performed and a kinked appendix removed, who, a week later, had an attack of renal colic, and was found to have tuberculosis of the right kidney.

A psoas abscess apparently cured by incision, injection of iodoform emulsion, and subsequent closure.

And a cyst of the antrum which was quite opaque on transillumination.

October 14th, 1912.

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A LECTURE

ON

THE TREATMENT OF BRONCHITIS IN THE STOUT AND ELDERLY.*

By HARRY CAMPBELL, M.D., B.S., F.R.C.P.,
Physician, West End Hospital for Nervous Diseases.

LADIES AND GENTLEMEN,—Stout elderly people often suffer from chronic bronchitis, and not infrequently they are carried off by it. It is bad enough to be stout and elderly, but to be stout, elderly, and bronchitic into the bargain, is to be sorely afflicted. The obese elderly bronchitic has long interested me, because his condition is so eminently amenable to common-sense treatment. I never see a sufferer of this kind without experiencing an impulse to explain to him how unnecessary most of his suffering is. Provided his kidneys are healthy, we can generally promise him a great amelioration of his condition—if not, indeed, a complete cure.

There are three main factors in the causation of bronchitis: (*a*) The inhalation of impure air, (*b*) a poisoned state of the blood, (*c*) feebleness of the circulation in the bronchial mucous membrane.

(*a*) With regard to the first factor let me remind you of the irritating effect on the conjunctiva of a thick London fog. This acts in exactly the same way on the mucous lining of the respiratory tract. Consider again the frequency of rhinitis among the inhabitants of large towns. All central Londoners appear to suffer from some degree of this affection, owing to the constant irritation to which their nasal mucous membrane is subjected. Hence the importance of securing a pure atmosphere for the bronchitic patient. To this end he should always breathe through the nose. If you have a patient suffering from a chronic lung trouble

* Delivered at the Polyclinic.

of any kind, one of the first things to do is to see that his nasal passages are free, so that the air before entering the lungs shall not only be warmed and moistened, but, what is still more important, filtered of any irritating particles it may contain. It is because the nasal mucous membrane bears the full brunt of this unfiltered air that rhinitis is so much more common than bronchitis. The importance of this protective function of the nasal passages in regard to the lungs is shown by the fact that the cure of a nasal obstruction sometimes actually leads to the disappearance of a chronic bronchitis.

(*b*) We have next to consider the influence of poisoned blood in the production of bronchitis This is much the most important causative factor. The hæmic poisons which produce inflammation of the respiratory passages may be taken into the body from without, or they may be autogenetic. Chronic alcoholism is an instance of the first form of poisoning; tissues which are saturated with alcohol are predisposed to inflammation. Autogenetic toxæmia may result either from bacterial agency or from vicious metabolism such as occurs in Bright's disease and diabetes, in both of which bronchitis is likely to occur.

A potent source of blood-poisoning, and one very important from the therapeutic standpoint, is that which results from intestinal sepsis. We have all heard of children who "cut their teeth with a cough." I take it that the explanation of this undoubted occurrence is this: The eruption of the teeth tends to disturb digestion; intestinal sepsis following, this begets toxæmia, which, in its turn, sets up bronchitis. I do not remember ever to have seen a child with so-called "consumption of the bowels" (giving rise to putrid, slimy, and, perhaps, blood-stained motions), in whom some degree of bronchitis was not present. As another instance of blood-poisoning causing bronchitis I may mention gout, which in the opinion of some is caused by the absorption of poisons from the alimentary tract. Bright's disease is a frequent cause of bronchitis. In all cases of bronchitis in the elderly and aged examine the urine. If the kidneys are diseased, we cannot hope to do more than alleviate the trouble.

How comes it that in toxæmic bronchitis the brunt of the toxic attack so often falls upon the bronchial mucous membrane? In seeking an answer to this question we must bear in mind that all the surfaces, internal and external, of the body are excretory: Poisons circulating in the tissues escape from every available surface—the skin, the kidneys, and the mucous membranes. We have abundant evidence that many poisons besides carbonic acid are eliminated from the respiratory tract: A person continues to exhale fumes of alcohol for some time after he has imbibed it. Everyone, again, knows how the smell of onions hangs about the breath, no matter how thoroughly the mouth has been rinsed out. This is due to the exhalation of volatile substances which have been absorbed into the blood. Sometimes the breath gives evidence of the elimination of autogenetic poisons, as in acidosis. It is very common, *e. g.*, for the breath to have an ethereal odour when the digestive organs are disturbed. My own sense of smell happens to be acutely sensitive to these odours, and I can testify to the frequency of ethereal exhalations in minor disturbances of health. Sometimes the gaseous poisons given off by the respiratory mucous membranes are offensive, even fœtid; a "bad breath" is by no means always the result of disease of the mouth, throat, or nose, but may be due to hæmic exhalations. And not only do poisons escape from the respiratory mucous membranes in the gaseous form; they may also pass out in the liquid form, in which case they are removed by expectoration.

In short, the respiratory tract affords an important channel for the elimination of poisons circulating in the blood. As a result of this elimination its mucous lining is liable to be irritated into inflammation.

(*c*) Let us now look at the third factor in the causation of bronchitis, *i. e.* sluggishness of the blood-flow through the bronchial membrane. You are familiar with the fact that in heart disease the patient is apt to suffer from bronchitis. This is largely on account of the sluggishness of the bronchial circulation. When for weeks and months together the blood has been flowing sluggishly through a tissue the vitality of the tissue suffers, and it is predisposed to inflammation. Now we know that stout people are prone to bronchitis. I think this is in large measure because in them the circulation is sluggish. Stoutness predisposes to sluggishness of circulation in three ways: (1) By producing or aggravating emphysema, which

hampers the circulation. All very stout people are emphysematous. They have high shoulders and expanded chests. This distension of the chest results, as I am always insisting, from the fact that in all cases of dyspnœa other than that due to mechanical obstruction of the larger respiratory passages, the inspiratory muscles act more powerfully than the expiratory ones. (2) Further, in the obese there is actual mechanical interference with the action of the heart by the copious deposit of fat about this organ. (3) Finally, there is the circumstance that stout people, by virtue of the excess of weight which they have to carry about with them, are debarred from taking a normal amount of exercise. Now exercise promotes a vigorous circulation, and the absence of it predisposes to circulatory sluggishness.

Whether or not I am right in my supposition that the liability of stout people to bronchitis is due to the sluggishness of the bronchial circulation, one thing is certain, *i. e.* that when we have to treat a stout bronchitic we should at once set vigorously to work to reduce his weight to the normal. I cannot exaggerate the importance of this. Unless this class of patient is vigorously handled he will keep stout to the end and die before his proper time. Your stout bronchitic does not take exercise, because he so easily gets out of breath, and the lack of exercise makes him stouter still: there is a vicious circle. Moreover, his breathing is hampered by his excessive fatness, and this interference with the proper aëration of the blood further promotes the laying on of fat.

We often experience a difficulty in getting our obese patients to take the proper steps for reducing their weight. They tell us they are naturally stout; Which among us is not familiar with the plea, "I am sure it is not what I eat, doctor." Now I readily admit that some people are naturally stout, and that you may do much actual harm by reducing their weight; but in forty-nine cases out of fifty the stoutness is self-induced, and can be successfully combated if only the patient will do what he is told. I do not remember to have seen a really stout postman or navvy. On the other hand, I rarely see a London policeman over thirty years of age who has not a comfortable waist. If a person can be got to take proper exercise and to reduce his daily allowance of food to physiological requirements, his excess of fat will generally disappear. Distrust the stout person who says he eats very little; this very little when totalled up generally turns out to be a great deal.

We now come to the question of treatment. Let us take the case of a stout patient suffering from *acute bronchitis*. We are called, let us say, to see a sixteen-stoned man of sixty years, in the throes of acute bronchitis. He is propped up in bed gasping for breath; his face is cyanosed and suffused with sweat, and his legs are œdematous. How are you going to treat him? First of all, you must give him cool fresh air to breathe. You wheel his bed or chair (frequently the latter) to the open window. I am quite sure that if I was suffocating from acute bronchitis I should crave to sit near an open window. Next you order a tube of oxygen to be at hand. There is no doubt that patients get comfort from oxygen; they can use it as their instinct directs. You apply a dozen leeches over the liver and encourage the flow of blood by subsequent hot fomentations. You order 5 gr. of calomel, and a mixture containing carbonate of ammonia and senega. As for the œdema, the most effective way of getting rid of it is by means of digitalis and theocin sodium acetate (5 gr. *ter die*).

As regards food it should in all cases of this kind be practically withheld altogether for a few days at least. I lay great stress on this. I know no more fitting inscription for a hospital devoted to diseases of the chest than this—"In all cases of acute dyspnœa *starve* the patient." Many lives are sacrificed through ignorance of this common-sense precept. Every particle of food which is absorbed goes to increase the production of carbonic acid, and it is the excess of this in the blood which causes the dyspnœa. You need not fear any ill-results from withholding food for days together. It is not easy to kill a patient by starvation. We know that professional fasters can go without food for forty, or even fifty days. Now these men are not obese. I see no reason why a very fat man should not live as long without food as a very fat pig, which can live for several months without food.

Some of you may remember the case quoted in works of forensic medicine of a pig which one evening made its bed on a straw-sta : in course of construction. The next day the men, all unconcious of its presence, came and piled up the straw over the creature, and no one knew what had become of it until some 150 days afterwards (I

think it was) when the stack was taken down. The pig was found alive, though of course much reduced in size. I believe that if a very fat man were to test his fasting powers they would be found greatly to exceed those of the professional fasters, who when they start to fast have not as a rule a superabundance of fat.

I say you need have no hesitation about withholding food for some days from the stout bronchitic patient who is in danger of suffocation. I am certain you should not give food to any patient who has acute dyspnœa, above all if he is obese. It is strange that the physician has so long overlooked the fact that every particle of food which is absorbed into the blood, and not laid down as tissue or run out as albumen or sugar, is katabolised, and causes an immediate increase in the production of carbonic acid, the excess of which in the blood is the essential cause of the dyspnœa. If you feel you *must* give the patient something to satisfy him or his friends let it be a little beef-tea. If you cannot persuade him to put up with this, then let him have a small portion of minced underdone lean meat. This is little likely to produce flatulence, which necessarily tends to aggravate the dyspnœa.

The patient may drink as much water as he likes, or he may have weak China tea. Unless he is a hard drinker, in which case the sudden reduction in the usual daily allowance of alcohol may induce delirium tremens, I advise you to give little or no alcohol

Let us now consider the treatment of *chronic bronchitis* in an obese subject. Bearing in mind the chief factors in the causation of bronchitis —irritation of the bronchial mucous membrane (either by the inhalation of impure air or the circulation of impure blood) and sluggishness of circulation, we are careful that the air the patient breathes shall be as pure as possible, and we endeavour to bring about a vigorous circulation of healthy blood throughout the body.

As regards climate, a pure, dry, warm air is the best. Cold air is not in itself bad for the lungs— it has rather a stimulating effect. The injurious effect of cold climate, especially a cold damp climate, on the respiratory mucous membrane depends upon its unfavourable effect on the skin. The skin excretes less actively in a cold damp than in a warm dry climate. Now the respiratory mucous membrane and the skin are to some extent complementary to one another, and if the action of the skin is checked, much of the poisonous material which is normally eliminated by it is got rid of by the respiratory membrane, which in this way may be irritated into inflammation. The bronchitic therefore does best in a warm, dry climate. A warm climate is, moreover, favourable to open windows and outdoor life, whereas a cold one tends to keep the patient in dusty, and perhaps stuffy rooms.

Our second object—the promotion of a vigorous circulation of healthy blood—is secured by common-sense means. We start at once to get the patient down to his normal weight. This is a very simple matter and does not require the services of a specialist. We have recourse to diabetic diet and prescribe a due amount of exercise. By these simple means we shall do much not only to stimulate the circulation, but to improve the quality of the blood. The digestive organs should receive special attention: the healthier they are the purer the blood. The withdrawal of starch and sugar is often followed by a considerable improvement in digestion and a reduction in the generation of poisons, which by their absorption into the blood exercise so injurious an effect upon the bronchial mucous membrane. Such starch as we may be disposed to allow should be chewed very thoroughly, so as to secure, as far as possible, its predigestion in the buccal cavity. In this way much of it may be converted into dextrines, and some of it even into maltose, before it reaches the stomach.

A favourite plan of mine is to order the patient a grain of grey powder and a teaspoonful of castor oil (in the form of a tasteless emulsion) every night for some months, or, instead of the oil, we may find it better to give half an ounce to one ounce of paraffin.

As regards expectorants, alkalies and ether are probably the best. The part of expectorants in the treatment of bronchitis is, however, a subsidiary one. Too great reliance has been placed upon them in the past to the neglect of common-sense measures. Bronchitis has never been cured by drugs, any more than dyspepsia has, but it often yields to common-sense treatment, and no variety more so than the chronic bronchitis of the elderly stout person. There is a danger that as we become more scientific we may neglect the more prosaic dictates of common-sense. Let us beware lest in our enthusiasm for modern scientific methods we neglect the obvious. This much I can assure you —you will do more for the obese bronchitic patient by reducing his weight, tending his digestion and regulating his exercise than by any other measures, scientific or otherwise.

October 21st, 1912.

DEMONSTRATIONS

ON

METHODS OF INVESTIGATING GASTRIC DISEASE.

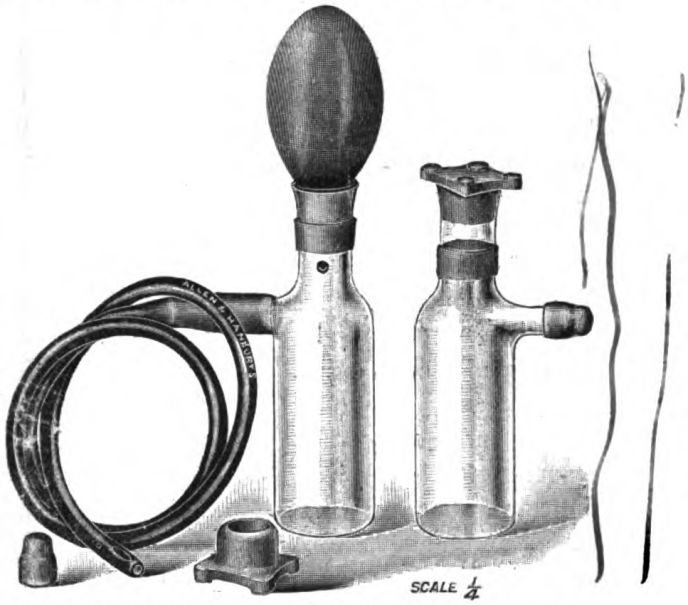
Delivered at St. Bartholomew's Hospital.

By W. LANGDON BROWN, M.D., F.R.C.P.,
Medical Registrar to the Hospital, and Physician to the Metropolitan Hospital.

II. The Chemical Examination of the Gastric Contents.

Gentlemen,—For the chemical examination of the gastric contents I always employ the Ewald test breakfast, consisting of one pint of tea without milk or sugar, and two ordinary slices of toast without butter. That might be considered a very unstimulating meal to the gastric juice, but recent work by Craven Moore shows that tea, contrary to our usual belief, is a stimulant to gastric secretion. The meal should be taken first thing in the morning, and some recommend that the stomach should be washed out before it. But I avoid this if I can, because when the patient has already had the stomach-tube passed that day, he is not so ready to have it passed a second time. And furthermore, I imagine that gastric secretion does not start under quite usual conditions just after the stomach-tube has been passed. If you want to test the motility of the stomach as well it is a good plan to give some food containing currants at four o'clock the previous afternoon. If currants appear in the gastric contents next morning there is undoubtedly deficient motility. You should not give acid or alkaline medicines for at least twenty-four hours before the test-meal, otherwise you may vitiate the result.

To withdraw the contents of the stomach I always employ Senoran's bottle, which is simple and efficient, and overcomes any difficulty in starting the syphonage. The patient should be sitting up with the head rather forward. The natural tendency is to throw the head back, but there is less difficulty if the head is tilted forwards. The tube is lubricated by being simply dipped into hot water and passed as quickly as possible, the patient being directed to swallow it, while you use the first finger of the left hand as a guide. The distance from the teeth to the stomach is generally $15\frac{3}{4}$ in., which is just under 40 cm. The tube is passed till the 45 cm. mark on it is level with the teeth. With the tube in position you squeeze the bulb and then, before releasing, put your thumb or finger over the little hole in the side of the bottle neck. There is then an efficient suction action in the tube, and the contents of the stomach are speedily extracted into the bottle. If you do not withdraw enough, repeat the squeeze, taking care to remove the thumb until you let go of the bulb, or else you will drive air into the stomach, which is uncomfortable. The bottle is provided with a rubber band to close the hole, and a rubber



The left-hand figure illustrates the bottle with the tube and bulb in position. The right-hand figure shows the bottle ready for carrying away. (Allen & Hanburys.)

stopper to make it portable. I have not had any difficulty with test-meals since I have used the Senoran bottle.

The contents of the bottle are filtered and some of the residue examined microscopically. If you find starch digestion very incomplete, that points to excess of acid, which stops the ptyalin of the saliva too soon. If the contents are frothy it suggests carcinoma. The presence of the long non-motile Oppler-Boas bacillus is said to be specially diagnostic of gastric carcinoma. Really it means there is no free hydrochloric acid, which is commonest in carcinoma. It is a lactic-acid-producing organism, and according to some it is the same as the Bulgarian bacillus, of soured-milk fame. The presence of sarcinæ is believed

to be a sign of dilatation of the stomach. No importance should be attached to the presence of yeast, which merely comes from the bread that the patient has eaten. Turning to the filtrate we proceed to examine for the presence of free hydrochloric acid. The best test for this is undoubtedly Gunzberg's. In an evaporating dish place a little phloroglucin, rather more vanillin, and dissolve in two or three drops of alcohol; add an equal number of drops from the test-meal and evaporate cautiously to dryness. A rose-red colour indicates the presence of free hydrochloric acid. It is now just beginning to appear. As a control I will repeat that with a test-meal which does not contain free hydrochloric acid, and you will see no colour appears. Now we proceed to estimate the amount of acid, both that which is free and that which has already entered into combination with protein, because that is physiologically active still. Merely estimating free acid does not tell you the total amount of HCl present. Chlorides are present in the stomach in three forms—free hydrochloric acid, HCl combined with protein, and mineral chlorides. Of these, the first two are physiologically active. Free hydrochloric acid can be best estimated by Töpfer's reagent (the dimethyl indicator), while we estimate the total amount of acid by using phenolphthalein as an indicator. Ten c.c. of the filtrate is placed into a beaker and diluted; two drops of Töpfer's reagent are added. If the gastric contents are acid they turn red. You now run in decinormal caustic soda until the red colour disappears, and is replaced by orange. In this case we find that 1·2 c.c. of decinormal caustic soda have been required to neutralise 10 c.c. of test-meal: that is to say, twelve would be required to neutralise 100. We say, therefore, hat the free acidity is represented by 12, or (as each c.c. of decinormal caustic soda is equivalent to ·00365 grm. of hydrochloric acid) ·0438 per cent. hydrochloric acid. Now I add a drop of phenolphthalein and go on. It will turn the solution red again as soon as the total acidity has been neutralised. We see this happens when 4·9 c.c. have been run in. That corresponds to a total acidity of ·178 per cent. hydrochloric acid. The normal total acidity is ·2 per cent., which corresponds to 5·5 c.c. of decinormal caustic soda when 10 c.c. of the filtrate are taken, and a little difference on one side or the other of that figure has no pathological significance. The estimation of the chlorides is rather an elaborate procedure; one specimen A of 10 c.c. of the test-meal is taken, neutralised by a saturated solution of sodium carbonate, and evaporated to dryness in a platinum dish; it is then charred, and the total chlorides are estimated in that. The other specimen B is evaporated and charred without previous neutralising, which will drive off the volatile chlorides. Specimen A gives us the total chlorides, and specimen B gives us the fixed or mineral chlorides. The difference between them gives us the volatile chloride, which corresponds, except for possible traces of ammonium chloride, to the hydrochloric acid. It is in doubtful cases of malignant disease that this more elaborate estimation is of service. For other purposes the estimation of the free and total acidity is generally sufficient.

	Gunzberg's test for free HCl.	Physiologically active HCl.	Mineral chlorides.	Total chlorides.	Ratio of active to mineral.
	Per cent.	Per cent.	Per cent.	Per cent.	
Normal	+	·2	·1	·3	$\frac{2}{1}\frac{0}{0}\frac{0}{0}$
Cancer	0	·10	·17	·18	$\frac{6}{1}\frac{0}{0}\frac{0}{0}$
	0	·175	·219	·394	$\frac{5}{1}\frac{0}{0}\frac{0}{0}$
Chronic gastritis	+	·15	·12	·36	$\frac{1}{1}\frac{2}{0}\frac{8}{0}$
Simple hyperchlorhydria	+	·34	·076	·416	$\frac{3}{1}\frac{1}{0}\frac{2}{0}$
Duodenal ulcer	+	·301	·155	·456	$\frac{1}{1}\frac{9}{0}\frac{3}{0}$

Now let me take some figures from cases illustrating what we learn from this chemical examination.

Whereas free hydrochloric acid, as shown by the Gunzberg test, is normally present, it is absent in nearly all cases of malignant disease of the stomach. In chronic gastritis there is usually some present, and of course it is present in simple hyperchlorhydria and duodenal ulcer. I have taken two cases of cancer as illustrating two different types. In the first case there is a great reduction of physiologically active hydrochloric acid. The mineral chlorides are scarcely increased at all, so the total comes out below the normal. In the last column is the ratio of active HCl to mineral chloride. Normally it should be greater than 1 but in carcinoma it will be much reduced. In the other case of carcinoma the loss of acidity is apparently due to neutralisation of the gastric juice by the alkaline secretions of the growth. Here, although

the physiologically active acid is not so markedly depressed, the ratio between it and the chlorides is definitely altered. As Graham points out, if the fixed chlorides exceed the physiologically active hydrochloric acid, that is highly suspicious of carcinoma. In chronic gastritis, beyond the fact that the physiologically active acid is lowered sometimes very considerably, there need not be any other very marked alteration ; we do not expect to find the ratio of active to mineral chorides less than 1. This is the principal diagnostic value of the estimation of chlorides. In both chronic gastritis and cancer of the stomach there may be absence of free hydrochloric acid ; in both the physiologically active acid may be reduced, but in chronic gastritis the physiologically active acid will exceed the mineral chlorides, whereas in carcinoma the proportions will be reversed. In simple hyperchlorhydria and duodenal ulcer there is a very marked increase in the physiologically active hydrochloric acid and the ratio is considerably greater than 1. From the test-meal alone it would not be easy to tell which of the examples given in my table was simple hyperchlorhydria and which duodenal ulcer, though mineral chlorides tend to be higher in the latter.

Occasionally it is desirable to examine the contents for lactic acid. Lactic acid in demonstrable amount in gastric contents is in favour of carcinoma. Extract about 10 c.c. of the test-meal with ether, shaking it well, then add the ethereal extract to water, and warm until the ether is evaporated. The lactic acid passes into the ethereal extract and is then transferred to the water. I test for its presence by Uffelmann's method ; 2 per cent. carbolic acid and one or two drops of a solution of ferric chloride gives a deep violet colour. Lactic acid turns this yellow.

The methods for the estimation of pepsin are not altogether satisfactory. Willcox's is the simplest ; it depends on measuring how little gastric juice is necessary in the gastric contents to curdle milk. There can be scarcely any doubt that pepsin and rennin are one and the same substance. Gastric juice is neutralised with an equal amount of decinormal caustic soda, and ·2, ·4, ·6, ·8, and 1 c.c. of the mixture added to 5 c.c. of milk in a series of tubes, which are kept warm at 40° for half an hour. Normally, clotting will occur in all tubes. If it does not occur, say, in the first two, it is evidence that the amount of pepsin is reduced. An objection to this method is that the preservatives often added to milk seem to interfere with the reaction. So far we have not learnt very much about the variations in pepsin, but it appears that in gastric ulcer, although there is an increased amount of hydrochloric acid, the pepsin is reduced. In gastric carcinoma and achylia gastrica it is usually entirely absent. In neuroses of the stomach the hydrochloric acid may be lowered, but the pepsin is normal. In any case the estimation of pepsin affords a clue to treatment. Obviously it is no use giving pepsin if normal curdling occurs, but it is useful where the secretion of the ferment is diminished.

I said that from the test-meal alone you could not diagnose very well between duodenal ulcer and simple hyperchlorhydria, but in the examination of the fæces for occult blood we have a very important guide. This is a simple enough test, and it is also valuable in the diagnosis of malignant disease of the alimentary tract, because by this method you will find blood in practically every specimen of the fæces. Small continuous losses of blood are typical of carcinoma of the alimentary tract. In simple ulcer you are more likely to meet with intermittent and larger losses of blood. If a patient has never had melæna but gives on three separate occasions a positive result with the test for occult blood, that patient has almost certainly malignant disease of the alimentary tract. I had a case quite lately in which the clinical features were very misleading, but this test was constantly positive ; the patient ultimately proved to have a new growth of the pylorus.

The test is performed as follows :

Take a small portion of fæces in a test-tube, add about 5 c.c. of water and boil thoroughly to destroy any vegetable oxidases, which would give a positive result, even in the absence of blood. A little benzidene is now added to some glacial acetic acid until a saturated solution is prepared. Ten drops of this are mixed with about three drops of boiled fæcal extract and then twenty drops of a 3 per cent. solution of hydrogen peroxide are added. If any blood is present in the fæces a blue colour will appear in about two minutes. A pale green colour is not enough to justify you in saying there is a positive reaction ; it must be a real blue. These are the usual quan-

tities, but exact measurements are not necessary. I will do the test now, and you see it is positive. I consider it one of the most useful additions to the chemical methods of examination of gastric diseases. But it is very important before employing this test that the patient should be put on a meat-free diet for three days, and that the bowels should be opened in between. A soap-and-water enema should not be used for this purpose, as soap tends to prevent the reaction. There is no objection to simple water enemata.

It is said that you should not allow the patient to have egg either, but on applying the test to egg itself I have failed to get a positive reaction; so I think you may allow eggs without vitiating the result. So if the patient is put on a diet of milk, eggs, vegetables, bread and butter for at least forty-eight hours, and the bowels have been opened properly each day in between, a blue colour with the benzidene test is positive evidence of small bleedings occurring into the alimentary tract.

Principles of Treatment in Dyspepsia.

I will now pass from the examination of the test-meal to certain principles which should underlie the treatment of achlorhydria and hyperchlorhydria respectively. For even when secondary to some more serious organic disease the principles of treatment of the gastric condition itself will be very much the same. In the treatment of atonic achlorhydric dyspepsia I need not dwell on such obvious points as attention to the teeth and bowels, but I will remind you that pyorrhœa and dental caries are more likely to cause trouble to those patients who have diminished hydrochloric acid than to the opposite type, because they are deprived of the antiseptic action of the gastric juice. In my experience it is in achlorhydria that the most careful scrutiny has to be given to the teeth. Secondly, we should naturally encourage the secretion of "appetite juice," by careful consideration of the patient's idiosyncrasies. It is a mistake to diet these people too rigidly. Thirdly, you should encourage the chemical stimulation of gastric juice by small quantities of meat extracts and the like at the beginning of the meal. It must not be a large plate of soup, which will distend the stomach and make them feel uncomfortable. But a little concentrated soup will act as a chemical stimulant which will help to cause secretion of gastric juice. Fourthly, carbohydrates should not be given in a soft form. And of course if they have bad teeth they will always tend to take their carbohydrates in as soft a form as they can. The starch will not be digested by the saliva if it is not masticated, but will tend to ferment in the stomach. Another important point which Cannon's observations with X rays have brought out is that if you give protein by itself it stays in the stomach some time, because the protein combines with the hydrochloric acid, and until there is free hydrochloric acid in the stomach the pyloric sphincter will not open. But carbohydrates given by themselves tend to pass out of the stomach very soon because they do not fix the hydrochloric acid, which, becoming free, opens the pyloric sphincter. But if you give protein and carbohydrate together, the normal onward progress of the carbohydrate is delayed, because the protein fixes the acid and prevents the sphincter from opening. The obvious deduction from this is that not only should we give these patients carbohydrates in a dry form, and therefore in a form requiring mastication, but we should give at least the main supply of proteins and carbohydrates respectively at different meals. It is a good plan to make the breakfast and the afternoon meal mainly carbohydrate, while getting in the greater part of protein at luncheon and dinner. In that way the protein does not interfere with the onward progress of carbohydrate, and there is not so much fermentation in the stomach. In passing we may note that the Continental breakfast conforms to this plan. Fifthly the achlorhydric patient cannot tolerate fats, because they tend to inhibit the secretion of gastric juice. Sixthly as to the drug treatment. As I said before it is a mistake to give these people alkalies after meals. If you do it is true you give temporary relief by neutralising the organic acids of fermentation which may be formed in the stomach, but you also prevent gastric digestion from proceeding. If you want to give these patients alkalies, which may indeed be quite useful, especially if there is excess of mucin, they should be given before meals. After meals the rational procedure will be to give hydrochloric acid. But many patients cannot tolerate a considerable dose of hydrochloric acid, and it has the obvious disadvantage of attacking the teeth. And so lately I have almost always employed betain chloride, which is sold under the name of acidol. This, when freshly dissolved, liberates nascent hydrochloric acid. It is put

up in pastilles containg doses equal to 7½ and 15 ♏ of hydrochloric acid respectively. These should be freshly dissolved in a wineglassful of water and drunk after meals. As acidol goes on liberating hydrochloric acid in the stomach for some time in this nascent form, you can get a much better effect from it than by giving ordinary hydrochloric acid. Lastly, we must remember it is the achlorhydric type of patient who is most likely to suffer from atonic dilatation. So look out for early signs of dilatation of the stomach and be prepared to treat it. The treatment of dilated stomach I shall deal with later.

Now let us consider the treatment of the opposite condition, that is to say, hyperchlorhydria. I am assuming that you have made a correct diagnosis, and that you have tried to exclude as far as possible all the other conditions which excite hyperchlorhydria. Here the administration of alkalies after meals will be a rational procedure, and no doubt the success of this treatment, indiscriminately employed as it often is, is due to the fact that so many of our dyspeptics are of the hyperchlorhydric group. It will be better to give them alkalies in the form which will not evolve a lot of carbonic acid gas. That limits us in the choice of an alkali; magnesia seems to be one of the best for this purpose. As bismuth plays an important part in reducing the hypersensitiveness of the gastric mucosa, it is well to combine it with the magnesia. This combination has the additional advantage that whereas bismuth is rather astringent the magnesia is relaxing. I generally start with an equal amount of each, say, bismuth carbonate 10 gr., heavy magnesia 10 gr., hydrocyanic acid 2 ♏, and chloroform water to the ounce. You will soon find out which requires to be altered. Equal quantities of magnesia and bismuth tend to be too relaxing, so probably more bismuth will have to be added afterwards. You can increase either drug up to 20 gr. I often prescribe the two drugs in separate bottles, telling the patient to regulate the dose according as he is constipated or relaxed. I think few things are better and simpler than the bismuth lozenges of the British Pharmacopœia, which were so warmly advocated by the late Sir William Roberts years ago; and they have the advantage of being portable. I direct the patient to suck one of these slowly when the pain comes on, swallowing with it as much saliva as he can, because this will help to neutralise the acid as well. Otherwise he may munch the lozenge quickly, so that the neutralising effect of the saliva will be lost. It might be thought that belladonna would be useful in the treatment of hyperchlorhydria, because it diminishes secretion generally. The usual experience with belladonna, however, has been distinctly disappointing. But Craven Moore has shown that if you give a small dose of belladonna, say $\frac{1}{100}$ gr. of atropin sulphate *before meals*, by itself it will diminish the secretion. It is no good adding belladonna to such a mixture as I have mentioned. It is open to other objections, however, and so I have not employed it to any great extent. Another factor which, according to Kaufman, is responsible for the pain of hyperchlorhydria, is the absence of gastric mucus, which is a normal protection to the stomach. It is probable that any success attending the empirical use of silver nitrate in gastric disorders is based on that fact. If you give these patients ¼ gr. of silver nitrate made up in a pill with kaolin for not more than five days you will excite a mild degree of gastric catarrh, which tends to antagonise the hyperchlorhydria. But obviously you must not continue it, because of the risk of producing argyrism.

Then as regards the influence of fats. We have seen that they have an inhibiting action on gastric secretion. And, therefore, it is reasonable to use them in the treatment of hyperchlorhydria. The patient has often found that out for himself. If not you may advocate the administration of olive oil or almond oil to these patient before meals. It sounds extremely unappetising, but I have known a number of patients who have obtained sufficient relief from it to make it worth suggesting. It may be given in quantities up to one ounce.

As to the dietetic principles in hyperchlorhydria, I have generally followed the plan laid down by Walter Broadbent some years ago, based upon Pavlov's work: and that is to give the food in a form which will not excite more secretion of gastric juice than can be helped. There are five stages in the diet. In my own practice I give the patient a paper with these stages written down, and I explain that if he is getting better he can pass from 1 to 5, and that if he is not so well he should work in the opposite direction. In the first stage, which is seldom required except when there is ulceration, he is only allowed milk and cream. In the second, bread and milk and other soft food. Thirdly, bread and butter and eggs. Broadbent says—although I have not found it so in all cases—that they are tolerated best in the form of buttered eggs, the butter still further diminishing the secretion of the gastric juice while the large amount of protein in the egg will tend to fix the hydrochloric acid. Fourthly, fish and vegetables, and lastly meat, while we entirely forbid the use of meat extracts of all kinds, because these stimulate the flow of gastric juice without giving anything for it to act upon. I do not put this table before you as providing hard and fast rules, but you will find that something of the sort is useful in the treatment of hyperchlorhydria, enabling the intelligent patient to modify his diet according to the condition of his malady.

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ON SYPHILIS OF THE NERVOUS SYSTEM IN INFANCY, CHILDHOOD, AND EARLY ADULT LIFE.

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(*Continued from page* 25.)

FROM THE CLINICAL STANDPOINT.

The following rough grouping will enable me to bring to your notice the more important clinical points. (1) The child or infant is brought for defective vision—amaurosis. In certain cases there may be other signs in the nervous system, *e. g.* spasticity or a cranial nerve paralysis. (2) Imbecility, idiocy and amentia. (3) Diplegic cases. (4) An epileptic group. (5) Meningitis and hydrocephalus. (6) Hemiplegia, cerebral thrombosis, hæmorrhage and embolism. Tumour and cranial nerve paralyses. (7) General paralysis and tabes. Cases resembling these metasyphilitic diseases, diffuse cerebro-spinal lesions, pseudo-tabes, Charcot's disease, (8) Spinal conditions. (9) The relation syphilis bears to other diseases of the nervous system.

(1) THE CASES WHERE AMAUROSIS IS THE MOST STRIKING FEATURE OF THE CASE.

My excuse for bringing this first case to your notice is that although the condition belongs rather to the province of the ophthalmic surgeon, the ocular condition may not be the only manifestation. Other parts of the nervous system may be involved. The retina and the optic nerve often show conspicuous changes; they are both parts of the nervous system, and in certain cases a cranial nerve may be implicated at the same time. I need hardly remind you, whether the case is an "eye" case or a "nervous" case, a thorough examination with the ophthalmoscope is of prime importance.

Let me first detail to you the main features of the case. We will then discuss the conditions with which we are likely to confound it.

CASE 1.—Harold C—, æt. 8 months.—*History.*—The parent sought advice because "she does not think the child sees properly. The eyes are constantly on the move."

Family history.—The patient is the sixth child. The first child died at two years and eight months with scarlet fever. The second child is alive and healthy. A miscarriage (twins) at seven months was followed by the birth of a fifth child (two and a half years) who is alive and is healthy. The mother is healthy.

Present state.—Examination of this child reveals the following: Well nourished but anæmic, fontanelle widely open; ? slight cranio-tabes. Dentition is delayed, and the epiphyses slightly enlarged. The child is apparently blind, or has only a very poor perception of light. There are no definite stigmata of the inherited disease. There are no signs in the nervous system beyond the fundus changes.

The ocular condition.—The eyes are constantly roving. The pupillary action to light appears to be normal. Under chloroform the media are hazy, but no definite opacities were seen. The discs are pale. The vessels seem smaller than normal. The retina is pale; there are a few irregularly scattered yellow spots. There is a pigmented area at the right macula.

The Wassermann reaction in the blood was positive. The case was seen in consultation with Mr. Stephenson, who said the child was suffering without doubt from "specific hyalitis."

Subsequent course of the case.—The child was put upon mercury and iodide, and when last seen (one year later) the following note was made: "Considerable improvement. Can walk about freely in the consulting room without any difficulty. Grasps objects in a natural way. The eyes are still constantly roving. There is an irregular strabismus. Pupils react normally to light."

The last examination of the fundus showed the media were much clearer than formerly. The dics are still pale. The vessels are still rather thready, and in the right fundus a pigmented area can now be definitely seen. Another paler area between it and the disc can also be seen.

The differential diagnosis of amaurosis in infants.—According to my colleague, Mr. Stephenson, there are five well-recognised causes of amaurosis in infants.

(1) *Specific hyalitis.*—The case I have just narrated belongs to this group. Summarised, the

main features of this condition are as follows: "Blindness is partial or complete, and is due to vitreous opacities. Fluctuation in vision is particularly suggestive. The underlying cause is a specific inflammation of the choroid or retina, or both. It corresponds to the secondary manifestations of the acquired disease and is nearly always bilateral. The leading ophthalmoscopic feature is hyalitis. The opacities are usually dust-like and the details of the fundus are obscured. Underlying choroidal or retinal lesions may be relatively insignificant and may only be determined when the vitreous has cleared. A dirty yellowish-white hue of the optic disc is especially characteristic. Evidence of past or present iritis is scanty. In babies a cloudy vitreous means "only one thing—syphilis" (Sydney Stephenson).

(2) The next condition is *amaurotic family dementia*, a condition first described by Mr. Warren Tay. This disease of the nervous system is practically confined to Jewish children and is associated with very typical fundus changes. It has a pathological anatomy peculiar to itself (Mott and Carlyll, Holmes). The disease has no relation to syphilis, although occasionally in isolated cases certain microscopic features, highly characteristic, occur (Gordon Holmes). I have one child under my care at the present moment. The Wassermann reaction is negative, and the child is the second member of the family to be afflicted in this way. Even in the most early stages of the disease a most cursory examination of the fundus with a dilated pupil cannot fail to show the large white area at the macula with the deep red central spot. From my own experience I would say that the fundus changes occur long before the motor disability appears, although I cannot tell you at what date they first appear. I am inclined to think the macula changes are present at birth or very shortly after.

(3) Occasionally *transitory amaurosis* occurs in the course of a basal meningitis (Gay); Mr. Stephenson has called this "*fleeting amaurosis.*" Although the patient is blind there are no ophthalmoscopic signs

(4) Mr. Stephenson and Dr. Ashby have described in infants another type under the title "*post-convulsive amaurosis.*" As the term implies this is associated with a convulsive seizure, and is due, presumably, to an extension of the nerve storm to the visual cortical centres, the period of discharge being followed by a period of exhaustion. Aphasic disturbances and hemiplegic phenomena may be accompaniments. There are no fundus changes.

(5) In another group of cases *blindness is due to previous attacks of meningitis* and is associated with definite post-papillitic atrophy. I have under my care at the present time a boy, æt. 7 years, who has markedly defective vision, and in whom I cannot find any other signs in the nervous system beyond a typical post-papillitic atrophy. The only illness this child has suffered from is measles, at 2 years, unaccompanied by meningeal symptoms.

It is most important to recognise this type of case. Early treatment will do much to restore useful if not perfect vision. The child's parent is perfectly healthy and gives nothing in her history which points to syphilis. The child is born after a healthy child. So far as we can judge certain elements of the visual apparatus alone have been involved. I do not think that the retina and optic nerve have entirely escaped. The main brunt has undoubtedly fallen on the vitreous. The positive Wassermann reaction substantiates our preconceived views as to the specific character of this type of lesion, and anti-specific treatment has been most beneficial.

(2) Imbecility, Idiocy, and Amentia.

It is difficult to accurately gauge the part which syphilis plays in producing defective mental intelligence in its various grades apart from well-recognised lesions of the nervous system. Whilst Thomsen and Leschly in their investigations of mental deficiency, epilepsy, blindness and deaf-mutism by the Wassermann reaction conclude syphilis does not play an important part in the production of these various defects, I am convinced in my own mind that it is a very important factor in producing the simpler grades, at any rate, of imbecility and amentia, and that, too, more often than is supposed. It is certain that statistics on the subject vary within wide limits. Dr. Mott quotes the following authorities: Binswanger 9·5 per cent., Ziehen 10 per cent., Bournville "exceedingly rare," Langdon Down 2 per cent., Shuttleworth 1 per cent., Telford Smith 3 per cent. and Brown 1 per cent. Dr. Ernest Jones, of Toronto, writes: "It is not yet definitely known what proportion of cases of ordinary

idiocy are due to syphilis (congenital), but it is certainly a considerable one, possibly amounting to 20 per cent. The Wassermann reaction may be found even when no other signs of congenital syphilis are evident, just as it may be in cases of parasyphilis in the adult."

Chislett also draws attention to this very important point. Dean finds syphilis responsible in 15·4 per cent. of cases, and, more important still, so far as this present communication is concerned, among his 330 patients only 13 showed evidence of syphilis apart from a positive Wassermann reaction. I have had the Wassermann reaction carried out in several cases of mental deficiency with and without organic signs in the nervous system (compare the list already given). In six out of eight of my patients who gave a positive reaction, mental deficiency as gauged by backwardness at school, delay in walking or talking, is a prominent feature of the case. Although the blood reaction in all these cases was positive, in three only, as already stated, were there signs which pointed without doubt to syphilis as the cause. I have not carried out any observations on the serum reaction of the parents of these children, but it is interesting to note that of Dean's cases among thirteen parents of children who gave a positive blood reaction nine parents also gave a positive reaction.

Bellingham-Smith and Woodforde have carried out similar investigations, but their results appear to only partially agree with those of Dean. Imbeciles of the Mongolian type rarely, in my experience, give a positive blood reaction. This appears to be in direct opposition to the view expressed by some, who hold, I believe, that syphilis plays an important *rôle* in this condition. I have only met with one such case. The infant, under one year, was under the care of my colleague, Dr. Chisholm, at the Children's Hospital.

She presented the typical Mongolian appearance and had a loud systolic bruit over the cardiac area. The infant was very small for her age. The post-mortem examination revealed no macroscopic evidence of syphilis. An incomplete interventricular septum existed and the child died presumably of inanition. The positive reaction was only noted a few days prior to death.

The dementia associated with Warren Tay's disease as stated above bears no relation to syphilis. The case I have under my care at the present time gave a negative reaction, as I expected it would. In the familial type of maculo-cerebral degeneration where mental decay is a prominent feature it is very questionable whether syphilis plays any part (Batten, Mayou, Oatman). The following are the details of a case in which delayed mental development, delayed walking and talking appear to comprise the main features of the case.

Case 2: Ellen G—, æt. 3 years and 7 months. *History.*—Brought by her mother because she is not like other children. She commenced to walk late, and is very backward in her talking. When she attempts to walk she is always falling about. As a baby she had measles and abscesses.

Family history.—The eldest son is æt. 24 years. Previous history good; married. The second, a daughter, æt. 22, is healthy. The third, a daughter, æt. 21, healthy. The fourth is a young man æt. 19; he is healthy. The fifth son, a young man æt. 17, had convulsions at the age of 8 months but has been healthy since. The sixth child, a boy, æt. 15, had "screaming attacks" at the age of 16 months, and was admitted as an in-patient to a hospital at that time for a fortnight. He was then said to be suffering from a "consumptive brain." He had a similar attack at the age of 14. He was again admitted to a hospital for three weeks. I have seen this boy; I cannot find any trace of syphilis or nerve disorder, but his blood has not been examined for the Wassermann reaction. The seventh child is a girl, æt. 14, and is one of twins. She is healthy. The other twin, a girl, died at the age of 3 months with "fits and some kind of skin disease." The ninth child is a healthy boy, æt. 11. The tenth pregnancy ended in a full-term child born dead. The eleventh child is a boy, æt. 8. He has been an in-patient at an infirmary for tuberculosis. I have examined this boy, but can find no evidence of active or quiescent tuberculosis. The twelfth child is a boy, æt. 7, who is healthy.

Present state.—A well-nourished child. Dolichocephalic. No obvious stigmata of the inherited disease. Tonsils and adenoids. She is very restless and very irritable. She is very backward in her talking. Her gait is uncertain, and she needs some support when she attempts to walk. The sphincters are not under control. Beyond slight inequality of the pupils (right > left), a slightly uncertain gait and an intermittent strabismus of

no fixed type, there are no organic signs in the nervous system. The plantar reflex, I may add, is of the infantile type. *The Wassermann reaction* of the blood was positive (June, 1911).

Subsequent progress of the case.—The child was lost sight of for nearly one year. The mother, in May, 1912, again attended with the child, and says the child "now tends to fall about still more when she attempts to walk." There can be no doubt that this is the case. The bruises on the child's head confirm her statements. She has had no fits or "fainting" attacks. She is so backward she has not yet been sent to school. Her vocabulary is very limited, being confined to simple words as "Dad," "Mum," and "Dear Boy." The sphincters are not yet under control. She can now walk without assistance, but her gait is rather "stamping." There is no tendency to fall or stagger that I can demonstrate. There is no tremor or ataxy of the cerebellar type. As before there is an irregular strabismus. The reflexes are brisk, and there is still a plantar response of the "infantile" type but not of the characteristic deliberate extension type of upper neurone (pyramidal) disease.

Remarks.—To my mind this case illustrates several interesting points. Were it not for the slightly stamping gait, the persistent plantar response of the infantile type and the tendency to fall about I should say, without hesitation, the case was one of simple amentia. It may be that a more extended observation will show some definite motor disability of the pyramidal tracts or a progressive lesion of the cerebellar type.

The inco-ordination which presumably is responsible for the constant tendency to fall about cannot be satisfactorily accounted for in the absence of other signs. The persistence of an indefinite type of plantar response might point to slight pyramidal involvement. There is, on the other hand, no demonstrable spasticity. The child's family history, while suspicious, is not conclusive. The number of miscarriages for so large a family is not abnormal. Although the child has tonsils and adenoids she is not deaf. I cannot correlate her backwardness with this defect. The positive Wassermann reaction of the blood is again the only evidence the child has congenital lues. In the absence of stigmata and other confirmatory evidence, it is fair comment to suppose this defective mental intelligence is not caused by syphilis, which is merely an accidental association. To my mind, however, it affords a good instance of the type of case I wish to draw attention to. Year by year evidence is accumulating which tends to show that in syphilis we have a poison which manifests itself in this way in childhood as well as in adult life. In this instance I have not had an opportunity to examine the cerebro-spinal fluid. I have not carried out a Wassermann reaction on the mother or any other member of the family. It is impossible to gauge the effect of treatment, the mother has been so "casual" in her attendance.

In cases of general paralysis occuring in the subjects of the inherited disease the mental states peculiar to that condition tend to manifest themselves at or about puberty (Case 8). On the other hand, defective mental intelligence may have been noted from a much earlier date.

In the case of Susan D— (Case 7), æt. 8 years, the child's disposition was noted to undergo alteration about four to five months prior to the date when she first came under observation. Although at first sight the case appeared to be one of general paralysis in a child, certain features, more especially the negative Wassermann reaction when applied to the cerebro-spinal fluid and the way the case responded to treatment, makes one think that the patient is not suffering from general paralysis. I shall refer at greater length to this patient later.

In the case of my little patient Daisy A— (Case 10), the defective mental development is very marked. She is only in the "baby" class at one of the elementary council schools. She is æt. 7 years. Her vocabulary is of the most rudimentary type and she does not even know her alphabet. When I first saw this patient I found she had, in addition to the points already mentioned, irregular, unequal and sluggish pupils. The consideration, too, of her case will be postponed until we are discussing general paralysis and allied conditions.

Backwardness in talking is a prominent feature in the child Annie E— (Case 4). Stanley F— (Case 5) is also very backward in all respects for his age. Harry H— (Case 11) is very backward for his age but is by no means an idiot or imbecile.

(3) The Diplegic Type.

There are at least two well-recognised types of diplegic patients—one in which the condition is noted at or shortly after birth; the other in which a distinct interval of apparent health precedes the manifestations of the signs and symptoms. In the present connection the former only concerns us. In the majority of patients mechanical factors prior to, or during, delivery are held to be responsible. In infants who die as the result of difficult labour, definite hæmorrhages (venous), consequent on engorgement for

the most, have been found in such situations as would give an anatomical explanation at any rate of the resulting palsy. The brain of a diplegic who has survived for many years shows certain residua, thickened membranes, areas of sclerosis, and porencephaly. At the present moment I have under my care a child who presents the typical picture we associate with this type of case. The delivery was a difficult one and instruments were used. The Wassermann reaction in the blood is negative, as one would expect.

There is a small group of patients in whom the spastic condition is present at the time of birth. Mechanical factors cannot to my mind be held to be responsible. The labour may be quite easy. "Fœtal encephalitis" is quoted by some as the cause of the condition. In rare instances the mother in the course of her pregnancy has suffered from some acute illness.

In syphilis I believe we have a potent factor for the production of such a syndrome. At least two of my cases, I think, are of this nature. One of them, it is true, is a twin, so that mechanical factors cannot be excluded. This child, however, has died within the last few days, and though I cannot give you a detailed account of the micro-pathology of the case, I can tell you that there were none of the usual findings we associate with the variety of diplegia as narrated above. It is also interesting to note that the other child, to use the mother's own words, "was exactly like it."

Dr. Charles Box, in an interesting paper entitled "The Syphilitic Factor in the Hemiplegias and Diplegias of Infancy and Childhood," thinks syphilis is certainly a factor in a certain proportion of the diplegias of ante-natal origin. It is more difficult to establish the part specific infection plays in the post-natal variety.

The following are the details of the first of my cases:

Case 3.—Marjorie B—, æt. 1 year and 3 months. *History.*—The child cannot sit up, has made no effort to talk, and is quite different from other children of her own age. She cannot see well, the mother thinks, but "is not blind." She is one of twins. The other child, who died after six days, was "exactly like it." No instruments were used. The labour was an easy one. Very shortly after birth the parent noted that the child was quite unlike other children in that the limbs were so stiff and "she lay all curled up with the arms and legs all bunched together." The child has never had any fits.

Family history.—First child, æt. 15 years, is healthy: no fits, snuffles or rashes; is the top boy in his class. The second, a male child, died of marasmus at 14 weeks, a full term child, but small: had no rashes or fits. The third child is a boy, æt. 12 years, and is healthy. The fourth, a full-term child, was born dead. The parent attributes this to a fall. The fifth child, a girl, is æt. 8 years, and is quite healthy. I have seen this child. The sixth confinement resulted in twins; the patient Marjorie survived; the other as already stated, died after six days.

State of patient (November, 1911).—Poorly nourished child, is intensely pale. The temporary teeth are much decayed. Small condylomata round the anus and vulva. Very restless and irritable. Heart and lungs healthy.

Nervous system.—All the limbs are very spastic, and a few athetoid movements of the upper limb are seen from time to time. The reflexes are very brisk, and there is a bilateral extensor response of the organic type. The eyes are constantly wandering and there is an irregular strabismus. The pupils are normal and equal. ? Vision defective, but she follows a light. The fundus: Both discs are pale and atrophic (primary atrophy) and there are no macula changes.

The Wassermann reaction of the blood is positive. The child was immediately placed on mercury and potassium iodide, which were continued until her admission to the hospital.

Subsequent course of the case.—In April, 1912, the spasticity was still well marked. The child cannot sit up except with assistance. Is still very pale. The squint persists. The eyes still wander in an aimless way. There are occasionally a few fine horizontal nystagmoid jerks Slight athetoid movements of the upper limbs are still seen. The skin lesions are very much improved. The mother thinks the child is "much improved and appears to see better."

The little girl was admitted to the hospital on May 20th, 1912, for *lumbar* puncture. This was performed, and a small quantity of clear fluid (tension increased) was withdrawn. Dr. Woodforde reported as follows:

"The fluid is clear. There is no coagulum, nor is there any deposit. Albumen amounts to ·005 per cent. (Aufrecht). Noguchi test shows a faint trace of globulin. Glucose is present. Films show no cells or organisms. Cultures are sterile, and the Wassermann reaction applied to the fluid is negative."

On May 21st the child's temperature had risen to 101·4° F. and the following note was made: "She does not know her mother. The rigidity of the limbs is increased, but there is no head-retraction. The lungs are normal." The next day the temperature rose to 106·2° F. and the child died at 11·45 a.m. She only vomited once.

At the post-mortem no definite lesion was found to account for the sudden termination of the case. There was no sign of cerebral or spinal meningitis. The brain showed much venous congestion but no macroscopic lesion was seen. No organ showed any sign of inherited syphilis to the naked eye. The body weighed 12 lb. 3 oz.

Remarks.—The family history to my mind affords no positive evidence. Instrumental injury can be excluded. The cutaneous manifestations were typical. The Wassermann reaction of the blood was positive prior to the time when treatment was instituted. The report on the cerebro-spinal fluid can be considered from two points of view: (1) owing to the persistent mercurial treatment the amount of albumen is slight and the lymphocytic excess is in abeyance, *i. e.* the case is recovering so far as we can judge from the cytological and chemical analysis; or (2) this is the kind of report we are to expect when we are dealing with this type of syphilitic invasion of the nervous system. Taken in conjunction with the report of the cerebro-spinal fluid from the next case I am inclined to the latter view. In syphilis of the nervous system and in metasyphilis the meningeal changes are always prominent and are in all probability responsible for the lymphocytic excess. Where, however, we are dealing only with faulty development or early decay of a pyramidal cell, only to take this one type of cell as an example, we should not expect to meet with a lymphocytic excess or excess of albumen. Unfortunately I have no report to give you of the presence or absence of choline or other products of nerve disintegration. Had there been excess of this substance it would, I presume, point to early decay of the neuron. Its absence, on the other hand, might reasonably be correlated with faulty development. The amount of albumen contained in, and the lymphocytosis of, the cerebro-spinal fluid in infants suffering from inherited syphilis has been made a special study of by Leo Baron. His main conclusion, if I interpret it correctly, is that the information to be gained by lumbar puncture may or may not be of service in diagnosis when we are dealing with inherited lues in the infant.

My second case of the diplegic type is a less perfect instance of this class of patient.

Case 4.—Annie E—, æt. 3 years.—This child was brought to hospital because a short time previously she had met with an injury to the head. Prior to this "she could walk and the eyes were not crossed." The parents are Jews and speak very little English. One was given to understand that prior to the injury the "child had been quite natural." From a careful consideration of the case I am convinced this must be erroneous.

Family history.—The patient is the fourth child in a family of five. The three elder children (æt. 9½, 8 and 5 years respectively) are said to be quite healthy. The youngest child, a little girl, æt. 5 months, is also healthy.

Present condition.—A well-nourished child with typical adenoid facies. She is not at all deaf but is backward for her age. When examined she is very irritable. She makes no attempt to talk and is clean in her habits. Although said to be suffering from the effects of a severe head injury received three weeks ago the child is quite placid when left alone and is in no pain. She is fully conscious and can sit up naturally. When made to walk, however, the gait is spastic, the limbs being rigid and the toes pointed. At first she could only walk with assistance. The motor disability is due to a slight spastic paraparesis with bilateral extensor response. On deviation of the eyes to the left there is deficient action of the external rectus muscle. The degree to which this can be demonstrated varies. On another occasion there was slight, but definite, disability of the right rectus. I do not think from repeated examinations that this is an alternating concomitant strabismus. The parent is quite certain the weakness of the left eye is recent, but, as already stated, it is difficult to know to what extent her statements can be relied upon. At one time I thought there was slight weakness of the left arm. There were no other signs in the nervous system. The fundus has been examined repeatedly but is quite normal. The *Wassermann reaction* of the blood is positive, and Dr. Woodforde gave the following report on the *cerebro-spinal fluid*: "The fluid is clear and there is no deposit. Albumen = 0·025 per cent. (Aufrecht). No blood. The Noguchi reaction shows a slight trace of globulin. Glucose is present in normal amount. There are a few lymphocytes—less than 5 per c.mm. Films show no organisms and cultures are sterile."

Remarks.—The child is still under my observation as an out-patient, and there is no change in her condition. I regard the case as one of slight spastic diplegia with deficiency of the left external rectus occurring in a child the subject of the inherited malady. Mental deficiency is a prominent feature. I do not think that adenoids, from which the child undoubtedly suffers, will satisfactorily account for her mental state. Injury, as it so often does, has accentuated the symptoms, and has drawn the attention of the parent to a condition which, in all probability, has existed since birth. The child has not active syphilis of the nervous system, but affords another instance of the way in which syphilis can produce a picture of faulty development of certain structures or a premature decay of certain neurons.

The case appears to me to be on all fours with the preceding one with this difference: the optic nerves have escaped and the pyramidal tracts in comparison have suffered in slight degree only. If we may exclude the presence of a trace of albumen and globulin, the cerebro-spinal fluid can be regarded as normal. Since mercury had not been administered prior to the time when lumbar puncture was performed, the count is a true indication of the result we should expect to find in such cases, and tends to bear out what I have already said when discussing the same point in the case of the child Marjorie K—. On the other hand,

I must again admit the case may be one of mild spastic diplegia accidentally associated with syphilis which is in no way responsible for the nervous lesion.

(4) Epilepsy.

There would appear to be little proof to substantiate the popular belief that syphilis in the parents is an important hereditary factor in epilepsy (H. Griffiths). I believe syphilis on rare occasions only is responsible for the production of epilepsy of the idiopathic type in the subject of the acquired disease. There is no doubt that it is so, but it is exceptional.

In syphilis, however, we have a potent factor, to a far greater extent than other poisons acting on a normal brain, for keeping up and accentuating this state. So great an authority as Fournier has given it as his opinion that epilepsy may be directly due to syphilis in its secondary stage, and that in those with inherited syphilis epilepsy of syphilitic origin is especially common.

Vascular lesions, dependent on syphilitic changes in the cerebral blood-vessels and cortical gummata, may be associated with Jacksonian epilepsy. The problem here is a different one. Fits dependent on such lesions are found in the subjects of the inherited as well as of the acquired malady. The epileptiform seizures which occur in the course of general paralysis in certain cases must not be lost sight of. If a systematic application of the Wassermann reaction were carried out in cases of what we at present are inclined to look upon as cases of simple idiopathic epilepsy, doubtless some interesting results might be arrived at. In the future we may be able to draw a distinction between epilepsy of the idiopathic type, occurring in brains previously damaged by syphilis, and epilepsy occurring in those in which syphilis plays no part. I have had the test carried out in eight cases of idiopathic epilepsy, major or minor, or combined, who have come under my care recently. In only one case was the reaction positive. The details are as follows:

Case 5.—Stanley F—, æt. 4 years. *History.*—Has suffered from fits since eighteen months of age. During the fit, which lasts for about one and a half to two hours, he "twitches all over and afterwards goes to sleep. The bladder and bowels are evacuated during the fit." He is a premature child (seven months). He is an only child. Labour was not unduly prolonged or difficult. Learned to walk late; never had snuffles or a rash. He is very backward. The mother is a well-developed woman; no miscarriages; the father is said to be healthy.

Present state.—The little boy is a very fragile-looking child: very "highly strung," and it was some time before I could gain his confidence to enable me to carry out a thorough examination of the nervous system. He is very pale and is poorly nourished. There are no definite stigmata. His speech is confined to simple words such as "Mumma" and "Dadda." He can walk by himself fairly well, and as confidence returned made rapid strides in this respect. At first he was constantly crying and preferred to be nursed on his mother's lap in preference to getting about like other children. He is clean in his habits. The submaxillary glands are enlarged, but not so the epitrochlear ones. The *Wassermann* reaction of the blood is positive.

The nervous system.—Beyond a bilateral extensor response of the organic type and a slight irregular and intermittent squint there were no signs of organic disease in the nervous system. The fundus is normal and there are no choroidal changes. The cerebro-spinal fluid has not been examined. The child has been treated with mercury, iodides, and bromides. There have been no more fits and there is a distinct improvement in the child's general condition.

Remarks.—In this case the persistent extensor response shows we are dealing with a brain damaged at some time by organic disease. Although the child presents no definite stigmata of the inherited disease, and I have no proof beyond the evidence of the positive Wassermann reaction of the blood, I am convinced the child has inherited syphilis, and that treatment with bromides and mercury will prove more efficacious than with bromides alone.

In the early months of life when fits indistinguishable from those of the idiopathic type are so common I believe they are more likely to occur in those who are the subject of inherited syphilis than in those who are not. Only a small percentage of such cases will present definite signs of syphilis in an unmistakable manner. But by suspecting syphilis, by having a Wassermann reaction carried out, and, when this test is positive, treating the child with bromides and mercury we shall benefit our little patients to a far greater, and in a more permanent way, than by merely treating them in the usual way with bromides and other methods aimed at improving their constitutional state.

From my own experience I would say that the major type of epilepsy is the one which is especially liable to be associated with the inherited syphilitic state. I have not come across any child in which syphilis and minor epilepsy coincided. Whereas so many epileptics become imbecile after a time, whilst others, who differ in no way from them so far as their fits are concerned, retain their normal mentality, it would be interesting to see if syphilis, especially masked syphilis, and therefore in all probability untreated syphilis, plays any part or determines in any way whether the individual will become imbecile after a time as a result of his repeated fits.

(*To be continued.*)

October 21st, 1912.

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A POST-GRADUATE DEMONSTRATION AT ST. BARTHOLOMEW'S HOSPITAL.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

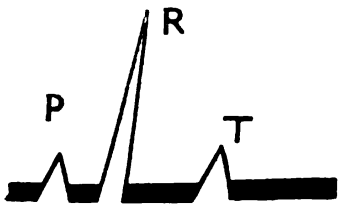
GENTLEMEN,—It happens that, partly owing to my having been away, and partly because my wards have been turned out, I have not many cases in the wards to show you. What I have are principally cases of heart disease of various kinds, and I thought it might be worth talking about some things which you may not have the chance of following out in the midst of your practice. In other words, I thought we might run over the points which are now being talked about concerning heart disease, such as tracings of the arterial and venous pulse, and so on. It is extremely interesting, although I cannot say it has yet led to any practical conclusion with regard to the treatment of heart disease, because it is largely English work. It originated under Dr. James Mackenzie, and is being carried on with great cleverness by Dr. Thomas Lewis, at University College Hospital. These two men have done as much as anybody in the world to elucidate heart disease in the last ten years.

Everybody knows that the contraction of the heart begins with the auricle and is continued from the auricle to the ventricle, but since we were taught that there has been a good deal more found out. And what has been found is, that not only does the contraction spread from the auricles to the ventricles, but that it spreads from a special spot in the heart. In cold-blooded animals, the hearts of which are less developed than those of man and not so complicated, there is a cavity called the sinus venosus, which is where the venous blood enters the cardiac apparatus. And in those hearts where the sinus venosus is fully established the contraction can be shown to start from the sinus

venosus: it is the point from which the impulse spreads to the rest of the heart. In the human heart there is no sinus venosus. But it has been found by experiment upon dogs' hearts that in the mammalian heart there are spots round the opening of the superior vena cava and inferior vena cava which correspond to the original sinus venosus of lower animals and are specially excitable. Secondly, there is a special differentiated tissue which leads from those excitable points about the entrance of the great veins down to a special spot, close to this opening which I show you on the fresh heart, the coronary sinus, and just below the fossa ovalis. And at that spot there is a collection of cardiac muscular tissue which is different from the ordinary cardiac muscular tissue. It differs from it in being more spindle-shaped, and you can sometimes see, even in the human heart, and better in the sheep's heart, that it is of somewhat lighter colour than the rest of the heart. The spot which I am now touching looked a different shade when I was dissecting it out, but the whole heart has now naturally become stained with blood. It is called the auriculo-ventricular node, and it is the spot from which runs down a special bundle of fibres. You are now just above the auriculo-ventricular valve on the right side, in front of the orifice of the coronary sinus and below the fossa ovalis—that is to say, you are on the septum of the auricles. Here is the septum of the ventricles below, and here I show you the auriculo-ventricular valve. The tissue which is specially differentiated in this way runs in a small longitudinal band through this tissue at the ring of the auriculo-ventricular valve, which is the fibrous septum of the heart, and then divides into two pieces, one of which goes to the right side of the septum of the ventricle and the other to the left side. It can be actually seen in many hearts as a yellow streak. So from this one point in the wall of the right auricle you can trace almost with the naked eye, but easily with the microscope, a bundle of fibres structurally differentiated from the rest of the cardiac tissue down on either side of the septum. This bundle of tissue, which is differentiated from the rest of that of the heart, is partly nervous and partly muscular. Its function had to be proved by physiological and pathological experiment. Physiologically that was done originally by Gaskell on the hearts of cold-blooded animals. He cut this bundle across, or clamped it and squeezed it with forceps so that it no longer acted as a conductor, and then showed that you could not send the stimulus through from auricle to ventricle. Other people have proved it in the case of dogs and other warm-blooded animals. And it has been proved pathologically in human hearts that the function of this bundle is to conduct impulses from the one to the other, and that when it is very badly damaged there is a stoppage between the two. The same has been proved also, and a great deal more has been found out, through the electro-cardiograms which are now being taken, and through the venous and radial tracings of James Mackenzie, who began telling us about it many years ago. You know what an ordinary sphygmogram of the radial pulse is. There is a systolic wave and a dicrotic. Nobody knows exactly what the dicrotic wave is, but it is supposed to represent closure of the semilunar valves. We need not, however, talk now about the meaning of the different waves. Mackenzie traced simultaneously the arterial pulse at the wrist and the venous pulse at the neck. In many people, if you look carefully, you can see at the root of the neck a double pulsation. This double pulse was noted long ago by Potain, but nobody made much use of it until Mackenzie began tracing it. The first of the two is presystolic in time, as can be seen by exactly comparing the synchronous events in a radial and a cervical tracing. That wave is produced by the auricular contraction. So here you have recorded the movements of auricular and ventricular systole separately, and it is by the study of those events and their variations that all the knowledge that is now possessed has been obtained. It is difficult to get the technique right, and wants practice. A good deal depends on where you put the cup on the neck of the patient. In some tracings the carotid wave comes out much bigger than the venous, when, by merely shifting the cup or the patient, the auricular becomes the larger. Another thing which has been done is to work on what is called the electrocardiograph. That is difficult for outsiders and those who are not physiologists to understand. You know that in an ordinary muscle nerve preparation, if you put a muscle in an electrical circuit and put the magnet needle within the influence of that circuit, when you send an impulse through that circuit so that the muscle contracts, the

magnetic needle turns aside one way or the other. The electro-cardiograph is founded on the same principle, but it works in the reverse way; you keep your magnetic poles constant, and the thing which does move is a fine wire which is part of the electric circuit. The circuit is made by putting the right hand of the patient and the leg on the opposite side in basins of warm salt water connected up with two electric poles. That brings the patient into the electrical circuit. The patient is kept perfectly still—so still indeed that the only muscular movement going on is that of the action of the heart. Then the current passes through the heart on its way from the arm to the leg, and at each contraction there are deflections of the very fine wire through which the current passes. This movement of the wire is photographed by a continual moving plate, and so there are certain curves

Fig. 1.



produced. Unless the patient keeps perfectly still he produces by stray muscular contractions other curves which destroy the value of the tracing. But if successful the cardiac contraction produces certain perfectly regular effects. Here is an example of an electro-cardiogram (Fig. 1). There is a wave produced by contraction of the auricle, and another set of deflections produced by contraction of the ventricles, and you read these things by noting the shapes which are produced by those contractions respectively. The auricle produces a little wave which is always called "P." In a regular heart the next thing is a very sudden tall wave called "R," and after an interval another wave, called "T," and the whole of that part of the curve, including "R" and "T," is produced by the ventricle. In the normal heart these waves vary a little according to the direction in which you take the current through it, which is termed the "lead." But in the usual lead these two parts are regular, and the three waves follow at regular intervals. In abnormal hearts the two parts, that is, "P" on the one hand and "R" + "T" on the other, are arranged quite irregularly, and it is from the irregularity of these curves that one infers the variations in the heart. Why the contraction of the heart should produce this series of curves nobody at present knows. Nobody knows what part of the ventricular contraction produces "R," or what part "T." There are, however, many theories, and I do not feel any doubt that in the immediate future much more will be made out in the interpretation of these signs, and much fresh knowledge about the cardiac movement gained.

I have two sorts of cases here to show you. Everybody has heard of "heart-block." This man is one case, and I shall show you another man, who is also an instance of it. In cases of heart-block the radial pulse is usually extremely slow, and that is the case here: this man's is about 28. The radial and cervical tracings are typical (Fig. 2).

The first thing you do is to measure from the marks at the end which show the simultaneous position of the pens. Measuring on the line of the radial pulse you mark off the beginning of several of the systolic waves. Then you transfer these measures to the line of the cervical tracing, but in each case you must allow for the interval of time—$\frac{1}{10}$ of a second—by which the pulse in the carotid precedes that in the radial. That is easily done from the time-marker, which gives $\frac{1}{5}$ sec intervals.

With that allowance you can mark out the points on the cervical tracing which correspond to the beginning of the radial pulse, and also the points corresponding to the dicrotic wave, which marks the end of systole.

In the present tracing you have no difficulty in seeing that there are certain waves in the cervical tracing which correspond to the radial pulse, and which must, therefore, represent the carotid pulse. These are always called "C" waves. They correspond to ventricular systole.

But when these are marked off you see (*a*) that there are many other waves which must have some other origin, and (*b*) that some of the carotid waves are double or are differently shaped from the others.

Here comes in the interest of the tracing. What do these extra waves and odd shapes mean? Now if you measure you will find (*a*) that the second set of waves marked "A" are equidistant from each

other, (*b*) that the same distance brings you to the double carotid wave, and the flat carotid wave, each marked "A + C." Evidently, therefore, the odd carotid waves are altered by being combined with one of the other waves. Thus you reach the conclusion that there are two sets of waves, each with its own rhythm running independently through the tracing. This second set of waves is produced by the auricle.

So you will see here is a condition of heart in which the auricle is beating at something like the usual rate, which is about 70 per minute, but

stand until you are accustomed to it, but shows the same thing.

This is another class of case which is very common, and is called auricular fibrillation. It is a pulse which goes very irregularly. But the characteristic of the cervical tracing is that it shows a venous pulse without any mark of the contraction of the auricle, and that has been called by some people a ventricular venous pulse, meaning that there is no evidence of separate contraction of the auricle (Fig. 3). For a long time nobody made out what that was, and at first it was thought the auricles

FIG. 2.

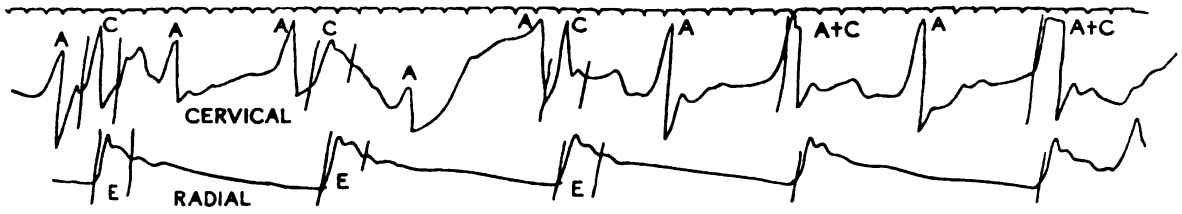
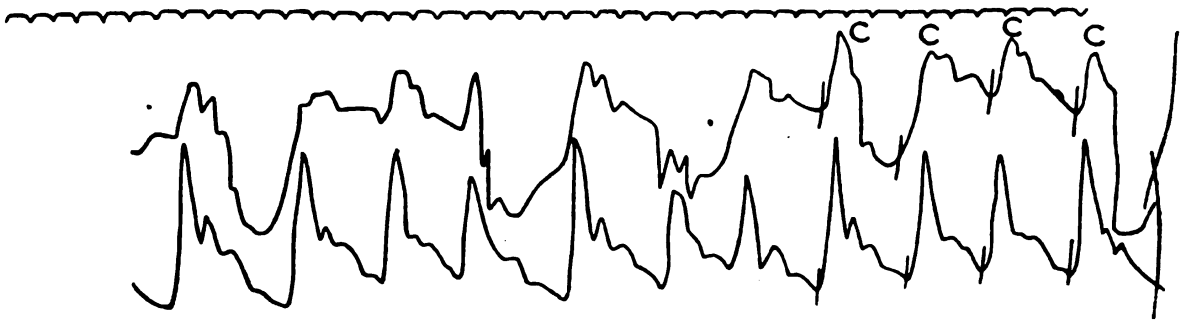


FIG. 3.



in which the ventricle seems to be going entirely on its own, and much more slowly: in this patient it is 28, and the auricle and the ventricle seem to have no communication with one another. Those are cases of heart-block, and it means that there is no conduction of impulses from the auricle to the ventricle, and that the contraction of the ventricle is not produced by contraction of the auricle, but is on its own. And it happens that when a ventricle is producing its own contraction it always contracts very slowly. An auricular contraction rate is usually about 70 per minute, but the ventricular contraction 28 or 30 per minute. You can trace it in these cases easily enough, and in some patients you can actually hear the auricular contraction in intervals between the ventricular. The electro-cardiogram is a little difficult to under-

were completely paralysed. And then it was found that in many of these cases the auricles were very hypertrophied, and they did not at all look as if they were paralysed. Then it was found experimentally that you produce a tracing like this drawing by faradising a dog's auricle, and that if you did so you started the auricle quivering or fibrillating, and it never contracted as a whole; the muscular fibres flickered as they do in a case of progressive muscular atrophy. Later the electro-cardiogram showed a tremulous vibration occurring through the whole cycle, but no "P" wave of auricular contraction. And later still Lewis saw the whole thing happen as an idiopathic disease in horses. It appears that horses have this particular form of cardiac irregularity. Lewis cut open a horse when the heart was still at work, the horse

having been shot through the brain, and he could actually see the process going on in the heart of the horse which we know must be going on in the human heart in these cases. The result of it is this: When the auricle is flickering like that, it does not send down its impulses at all regularly, and it appears that it is only occasionally that an impulse gets down when that is the condition of things. There is no disease of the conducting tissue between the auricle and the ventricle as there is in cases of heart-block, so that if the impulse is strong enough it can get down to the ventricle. As the auricle is flickering in an irregular way those impulses are irregular, both as to time and force, or at any rate are inducing contractions which are irregular in force. And in that way you have a curve of ventricular systole, which obeys no rule of force or of time. It is irregular in both those respects. Those cases sometimes occur in the young, sometimes in the old.

When you realise the meaning of it you might think it was a very fatal disease. But it is not. These cases sometimes go on for years. Sometimes patients with this condition die of general exhaustion of the heart. In old people the condition appears to make very little difference provided the conducting tissue is all right and impulses can get down to the ventricle. You have probably seen old people whose hearts are utterly irregular and yet who show no definite sign of cardiac failure. I had an instance the other day. An old clergyman came to me with the story that he had influenza, and since then he had sometimes been feeling dreadfully bad, so that he had to abstain from taking his service. But between those times he felt comfortable. I found he had a heart which was beating without any rhyme or reason: it was very irregular. And when you first see a man, æt. 50 or 60 years, with a heart which is not observing any rhythm you think that man must be dreadfully ill. But when I came to examine him I found his legs were not swollen, the liver was not enlarged, and his lungs were free. I asked him if he could produce the condition by going uphill and he replied that he could go uphill quite well. When you hear a statement like that from a man it is clear that his heart cannot be failing, and he may go about his work and not show any signs of cardiac failure. In these old people I do not think it matters to them very much so long as their condition is all right: they are really more frightened than hurt. I had some difficulty in persuading this clergyman that he was not as bad as he thought himself. The same thing may be said in cases such as I have no doubt you have seen yourselves, in which comparatively young people have been under your care for ordinary rheumatic mitral conditions with similar hearts, and when they left your care you thought they were going to die in a month, but they have not. I have known many such cases. The condition in the elderly is not rheumatic as a rule; if it is valvular at all—which often it is not—it is atheromatous. Often there is no murmur at all. In the younger cases there is generally a murmur. But in elderly people there is often no sign of mitral disease. It is a more serious condition in the younger patient than in the older. No drug seems to affect it at all in the old, but in the young digitalis seems to produce benefit. The doses must be large, and although this drug does not make the auricle contract it produces a steadier contraction of the ventricle. The explanation of that is much more difficult.

I hope I have not told you things all of which you knew before; I thought we would speak of them because they so absolutely revolutionise the ideas of the working of the heart which we acquired when we were learning physiology. Of all the remarkable bits of work which I know of, this by James Mackenzie is one of the best.

Lastly I show you two cases of exophthalmic goitre treated by thyroidectomy of one lobe at least. The first is a woman of middle age who was here in 1908. At that period she had exophthalmic goitre, and was incapacitated from earning her living. After treating her ineffectually for several months we operated upon her and removed one lobe of the thyroid, the other side being left alone. She is now very much better, and she has gone back to her work—hard work, namely, ironing in a laundry. There is a little exophthalmos on the right side, but her pulse is a good one, not very quick and quite regular. I feel sure the operation she had was the cause of her improvement.

This next patient is a young woman who shows a case from the other side. She had not had the condition so long as the other patient. She came to us two or three years ago, and then had some

exophthalmos—not a very large goitre, but a great deal of palpitation. She could not carry on her work. On her, too, we operated. We tied nearly all the arteries of both sides, one after the other, and then the whole of the left side of the thyroid gland was taken out. That was done more than a year ago, yet I am sorry to say she is not yet fit to earn her living, and never has been. The amount of palpitation is as great as it ever was. I do not know when a case of this nature is going to improve after operation and when it is not, and until we know the features which indicate that, operation seems to me to be risky. At the present time it appears to me that the cases usually cured by operation are the early cases, which after all can usually be cured, or often cured, without.

The last patient is a young woman with exophthalmic goitre, who has very marked pulsation of the pulmonary artery. It sometimes puzzles people to know what that pulsation is. It is in the first or second left interspace. We felt it must be the pulmonary artery by the position of it, and on examination of it by means of the X-ray screen we saw that it is synchronous with the systole of the heart; in other words, this part expands during the heart's contraction. It is a very expansile dilated pulmonary artery.

October 28th, 1912.

Use of Lactic Acid Bacilli in Pulmonary Tuberculosis.—P. C. Bartlett and C. V. Murphy state that every clinician who has concerned himself extensively with the care of tuberculous patients has been impressed with the prostrating effect of the obstinate diarrhœa, alternating very often with intervals of constipation, which is a not infrequent complication of well-established or advanced pulmonary tuberculosis. That the large percentage of these cases are functional only has been indicated by a somewhat extensive study of the fæces in the laboratories of the Rutland State Sanatorium, and their toxic origin is strongly suggested. It is in connection with this type of individual, as well as in cases of actual tuberculous enteritis, that the employment of lactic acid soured milk has afforded the maximum benefit, both as a dietary measure in support of energy and in tissue repair, and as an agent favourably modifying the symptoms and controlling the diarrhœa. Its range of usefulness extends over almost the entire field of gastro-intestinal affections encountered in connection with sanatorium work. — *Med. Record*, vol. lxxxii, No. 12.

ON METHODS OF EXAMINATION OF OPHTHALMIC CASES.

At St. Bartholomew's Hospital.

By M. L. HEPBURN, M.D., B.S., F.R.C.S.,
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One of the chief difficulties in dealing with eye cases in general practice is the inability to arrive at a correct diagnosis. This is due to the fact that the methods of examination are so different from those employed in other branches of medicine and surgery, and in the absence of any special training no definite system of investigation is present in the mind when approaching a case, and under these circumstances mistakes are only too likely to occur. I shall therefore lay stress on the methods of examination in showing the following cases.

The first case I show you is a type of conjunctivitis which is commonly met with, but must be accurately diagnosed in order to be treated successfully. General practitioners are expected to cure conjunctivitis, but they must be able to recognise one or two different types, and this case, which represents the form called angular, is one such type. It is due to a special organism, the diplo-bacillus, which is readily demonstrated from the conjunctival secretion, and you will notice that there is very little affection of the bulbar conjunctiva, which is almost as white as normal. In any case, whatever vascular injection there is is not very great, is confined to the external and internal canthi, and although so slight, tends to spread over on to the skin at these points, while all along the inner edge of the lower lid there is a certain amount of congestion of the conjunctiva. This patient complains of a good deal of itching, very characteristic of this type, while at the same time there is very little discharge.

It is distinguished from catarrhal conjunctivitis in that the latter shows very marked congestion in both ocular and palpebral conjunctiva so that the whole eye looks a brilliant red colour. There is a good deal of discharge, some of which sticks to the lashes and forms crusts. This form does not

usually extend over on to the skin of the lids, and by adopting ordinary methods of cleanliness will get well in about a fortnight; not so the angular type, which is often very chronic, and requires the application of some form of zinc lotion in order to effect a cure. The strength generally used in hospital work is two grains to the ounce, but I should always advise you to begin with one grain to the ounce in private practice, as patients often complain of stinging if the stronger preparation is used. In some cases it is difficult to distinguish between circum-corneal injection (which is always a sign of extreme importance) and conjunctivitis. In such cases, one of which I show you here, there is a distinct band of comparatively white conjunctiva at the equator of the eyeball between the redness belonging to the conjunctiva and that of the cornea; besides which the latter is more vivid in colour and on close inspection is seen to be formed by the distension of a deeper set of vessels. If a conjunctivitis of any kind shows no signs of material improvement in a few weeks, it is well to paint the lids with protargol 30 per cent., or silver nitrate ten grains to the ounce, once or twice, separated by an interval of a day or two, but in all probability the delayed improvement is due to some undiscovered cause of the conjunctivitis such as I show you in the next case, and a thoroughly systematic inspection of the lids must be made.

Here we have a man who has suffered for months from a very chronic form of conjunctivitis, and on careful examination of his lids by the methods I shall show you, you will notice the conjunctival tissue has a thickened fibrous and contracted appearance with no trace of normal conjunctival blood-vessels visible anywhere. On eversion of the upper lid there are to be seen beneath the conjunctiva some small white raised spots. This is a case of granular ophthalmia or trachoma, and at once you know that the course of the case must be very prolonged, no matter what method of treatment be adopted.. This man has been treated by the application of nitrate of silver, and he has so far improved that I now propose to apply copper stick twice a week to complete the cure.

The next case I show you is that of a child, æt. about 18 months, with a hypopyon ulcer—an extremely dangerous type of corneal inflammation. You will observe about the centre of the cornea a rounded fluffy-looking ulcer with ill-defined edges, and on depressing the lower lid there is seen a large amount of pus in the anterior chamber which has been exuded from the iris as a result of toxic infection from the surface. You will also notice that although the child looks ill it does not seem to be conscious of any inconvenience from the bright light but keeps the eye open, nor does he shrink from any manipulation necessary for examination. This is unusual with children, who generally keep their eyes tightly closed when an ulcer on the cornea is present. It is a significant and dangerous sign, showing that the sensitive nerve-endings have been destroyed by the virulence of the organism which attacks the cornea at this spot, thus demonstrating a common experience in ophthalmology, namely that some of the most severe affections of the eye have to be definitely looked for and are often accompanied by very few subjective signs on the part of the patient. This ulcer must be treated vigorously or the eye will be lost in a few days. The ulcer must be cauterised deeply with the actual cautery, short of perforation; atropine must be instilled and attention paid to the general health by suitable food, tonics, and stimulants.

There are three ways of examining a cornea in order to determine the extent to which it is involved in any inflammatory process. One is by observing the reflection of a window on the cornea as seen in the different positions of the eye; if at any part the reflex is hazy or uneven there is evidence of affection of the cornea at this spot. The case I now show you is one of interstitial keratitis where the whole cornea is involved and an inflammatory exudation has taken place into the deeper layers, giving rise to what is termed "ground-glass" appearance. You ascertain that the surface is not destroyed by staining with a drop of fluorescein solution 2 per cent when you notice no green stain is produced, showing that the epithelium is intact. This constitutes the second way of examining the cornea and is an extremely useful one.

Here is another case of ulcer near the limbus on the lower inner side which is giving us some anxiety, because it is a deep one and has lasted a long time, while it shows little inclination to heal at present and may be extending. To determine this latter point we stain with fluorescein, and you see a bright greenish coloration where the epithelium is denuded, as well as some infiltration in the

deeper layers. You can thus state definitely that this ulcer has not healed, and perhaps the lids, which you notice to be inflamed, may be the cause of continual reinfection. If this be so, paint the lids after eversion with protargol or nitrate of silver, wash away all superfluous fluid after painting with a swab of cotton-wool by dabbing it on the lid, but do not neutralise with salt solution, as no harm can come to the ulcer from deposit of silver if reasonable care be used.

The next case is an extremely rare one and appears as an infiltration about half the breadth of the cornea, which starts from the limbus and advances slowly on to the cornea by a sinuous undermined edge. It will eventually, if left untreated, extend over the whole of the cornea without showing any tendency to perforate. It is known as a Mooren's ulcer, but this is an unnecessary refinement in diagnosis, and all that you need to remember is that it is likely to advance, and you must also decide whether it is actually proceeding at the present time. This may be determined by staining with fluorescein, and you see that the ulcers, which are present in both eyes, are advancing at their sinuous border. This must be treated with vigour, though the methods adopted are not always very encouraging. Cauterisation has been tried and is the old form of treatment, but only temporary improvement generally follows. We now treat such ulcers with zinc ionisation, and the results are undoubtedly better and prevent the recurrences which were so common under the older methods.

Cases of episcleritis, one of which I now show you, are often difficult to diagnose correctly, but a decision on the question is of extreme importance seeing that the condition is one that runs a chronic course and shows an obstinate resistance to many forms of treatment. The most likely error is to mistake it for a case of phlyctenular conjunctivitis which is of a comparatively trivial nature, and under appropriate remedies and hygenic conditions can be fairly easily cured. The latter is more common in childhood, while it is rare in adults. But the converse is true as regards episcleritis, so that the point for you to remember is that if a small inflammatory focus appears near the limbus in an adult which looks like a phlyctenule it is more likely to be episcleritis than this form of conjunctivitis, and a guarded prognosis must be given accordingly.

The next case I am showing you is one of concomitant convergent strabismus and I will briefly illustrate the methods of examination. The first point to decide is whether a child really squints or whether the deformity is merely apparent. Direct the child to look at any object about two feet from the face directly in front and then decide which is the squinting eye—in this case the right one. Having done this we now know that the opposite eye, the left, is the squinting eye and is not fixing the object. On covering the right eye you notice the left makes a movement outward in order to fix the object. This observation decides the presence of a real squint, for if the left eye had been fixing before, though in an apparently convergent position, no movement would have taken place in the squinting eye. The next point is to determine whether the squint is paralytic or concomitant. If you look behind my hand, which is covering the right eye, you will see that the originally fixing eye is now squinting to the same extent as the left one was before; thus the secondary deviation is equal to the primary which is characteristic of concomitant squint. In the case of paralytic squint the secondary deviation is greater than the primary, but such a squint is generally seen in adults, and we are greatly assisted in our diagnosis by testing the movements of the various muscles in the affected eye and by the history of the onset of diplopia.

All these points have to be settled when any case of squint presents itself and before any line of treatment is embarked on.

The next case I show you is a case of dislocated lens complicated by a band-shaped opacity in the cornea, and you notice first of all that the anterior chamber is deeper than normal. Now the anterior chamber is deep in three conditions: first, where the lens is absent or dislocated; second, in myopia; third, as a sequel to old-standing irido-cyclitis. We find that this is a case of dislocated lens because on movement of the eyeball the iris is tremulous, thus showing that the normal support of the iris is absent. The actual position of the lens may often be demonstrated in the dark room by reflected light as in looking for lens opacities, but one thing is certain—that in this case the lens is not in its usual position, and the accident must seriously interfere with accurate vision. I have here two cases of cyclitis and they are both in children (a boy and a

girl æt. about 10 years). Cyclitis is often very difficult to diagnose, and the only sign of its presence is the existence of small dots of keratitis punctata on the back of the cornea, which by examining with a magnifying lens are seen as round well-defined spots about the size of a pin's point dotted all over the cornea, but by no means always in the form of a cone so graphically described in text-books. This latter arrangement only occurs in the more acute cases when the individual spots are much larger, and where other signs are present which warn us of the serious nature of the affection we are dealing with. Although the extenal signs of cyclitis are the same as those for iritis with the addition of the spots of keratitis punctata, still, this is by no means always the case, and a very large proportion come on very insidiously and become well advanced before the condition is recognised owing to the absence of any characteristic sign.

On close questioning, periodical attacks of "red-eyes," passing off in the course of a few days, may be the only indication of there having been anything going on of an inflammatory nature, and the only reliable evidence of the cyclitis is the presence of keratitis punctata, which must be definitely looked for. It is well, therefore, to get into the habit of examining a cornea by oblique illumination and a magnifying lens, in the manner I am now illustrating, in any cases where you are in doubt about an eye which shows signs of inflammation a little out of the ordinary; and this constitutes the third way of examining a cornea. The girl is getting well, but you will notice the boy has a complete cataract in one eye as the result of the cyclitis, owing to interference with the nutrition of the lens due to inflammation in the ciliary body, which is its chief source of nourishment. I can get no history about it except some slight attacks of redness occasionally which were regarded as of no consequence, but the presence of very fine spots of keratitis punctata, which are pigmented, confirms the diagnosis. It is also of the first importance in deciding on the line of treatment. I should hesitate to operate on this eye, partly because he has a normal-sighted eye on the other side, and partly because a traumatism might light up the cyclitis again, which is now quiescent and old, but worse still, the inflammation might extend to the other eye and destroy the sight of that one also. I should therefore advise the mother not to have the cataract removed unless these risks could be proved justifiable.

October 28th, 1912.

ON SYPHILIS OF THE NERVOUS SYSTEM IN INFANCY, CHILDHOOD, AND EARLY ADULT LIFE.

By SYDNEY A. OWEN, M.D., M.R.C.P.,
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(*Continued from page* 48.)

(5) MENINGITIS.

(A) A well-defined type of syphilitic meningitis occurs in infants and young children. It is both basal and vertical in its anatomical distribution. From the clinical standpoint it resembles the more familiar post-basic type. The subjects may, or may not, show definite signs of inherited syphilis. The first eighteen months is the period in which it most frequently manifests itself for the first time. The onset is acute or subacute, with cerebral manifestations; strabismus, convulsions, vomiting, head-retraction and bulged fontanelle. The child rapidly develops spasticity of the limbs. In most cases a period intervenes between birth and the onset of the disease during which an unobservant parent may not detect anything amiss. In other cases there is a history of defective vision from birth with inability to sit up or even hold up the head. The course is very chronic. I am inclined to the view that a small percentage of so-called "recovery cases" of post-basal meningitis, prior to the time when lumbar puncture was infrequent, may have been of this type. As in the post-basic variety, hydrocephalus is a prominent feature. Thickened membranes, atrophied cortex, and enlarged ventricles are the three main features of the gross morbid anatomy (Batten). Microscopically the meninges show small-celled infiltration, plasma-cells are present, the cortical cells are atrophied or absent, and the blood-vessels show characteristic changes (endarteritis).

Lumbar puncture, in the acute stage at any rate, provides us with a means of differentiating the condition from other forms of meningitis. The lymphocyte count may be very high (92 per cent., Batten). In tubercular meningitis with excess of

lymphocytes a careful search may reveal bacilli and inoculation experiments would be conclusive. The later stages of certain other forms of acute meningitis are also associated with a lymphocytic excess. In their early stages, however, a polynuclear excess preponderates, and at this stage the specific organism is usually present. A high opsonic index is in favour of the epidemic disease. In all forms of acute meningeal infection the albumen and globulin count is raised and in tubercular meningitis the quantity of reducing substance is increased.

Of this type just described I have no recent instance to bring before you. I have suspected it in several cases but none with positive evidence can be brought forward.

(B) In the adult the subject of the acquired disease, a basal meningitis within the first eighteen months of infection is by no means infrequent. Dr. Mott describes a typical case in a young man, ? age, occurring within three months, of a soft sore, which had only been treated locally with iodoform. His patient suffered with diplopia and headache, and was found to have weakness of an ocular muscle, with a low grade of papillitis. Under mercurial treatment the symptoms rapidly disappeared. I have had under my care a child whose case appears to closely resemble the case just described. Whether the child is the subject of the acquired disease or the inherited I do not know. I am inclined to the former, and I think this is the only instance of the acquired type in my series. I can find no trace nor can I obtain any history pointing to a sore. The child presents no trace of stigmata. The family history is good, and the only other child is healthy. The Wassermann reaction of the blood is positive.

CASE 6.—Amelia D—, æt. 7 years. *History.*—Four weeks ago her illness commenced with attacks of vomiting, independent of food, violent frontal headaches, and difficulty in walking. Squint with double vision fourteen days. Pain in the back of the neck. Some cough, and the child is wasting.

Previous illnesses.—Measles and an ear discharge which lasted two months, but subsided without any definite treatment. The ear has not discharged for two months.

The child was admitted to the Queen's Hospital in May, 1911, under the care of Dr. Neave, *when the following note was made:* "The child is well nourished but pallid. There are no signs of disease except in the nervous system.

The nervous system.—Patient is quite conscious and complains of frontal headache on the right side. The right sixth nerve is paresed. The voluntary movements of the left face are not so extensive as those of the right (?). The tongue on protrusion deviates a little to the right (?). Her speech is normal and there is no nystagmus. The right knee jerk is not so easily obtained as the left, and the right abdominal reflex is not so brisk as the left. The plantar reflexes are normal.

Von Pirquet's reaction was negative. *The Wassermann reaction* applied to the blood was positive but negative for the cerebro-spinal fluid.

Lumbar puncture gave the following result (Dr. Woodforde): The fluid is clear and there is a small cloud of albumen. There is a trace of globulin. The lymphocytes amount to 10 per c.mm. Reducing substance is present. Cultures are sterile, and no organisms or polynuclears were seen. *A blood-count* gave the following: *Reds*, 4,500,000; *whites*, 16,000; *polymorphonuclears*, 73 per cent.; *lymphocytes*, 26 per cent; *eosinophiles*, 1 per cent.

The fundus.—The following note was made by Mr. Sydney Stephenson on May 25th, just three weeks after the child was admitted. "Both discs are recovering from papillitis, which is not of the choked disc variety. There are several small hæmorrhages on the right optic disc. The swelling does not exceed one dioptre. The appearances are consistent with cerebral abscess, tumour, or a basic meningitis."

The temperature chart showed slight irregular fever, 99° to 100° F. The pulse varied from 80 to 100. On June 9th she weighed 3 st. 5 lb. 6½ oz.—a gain of 5 lb. in less than a month.

The child was treated throughout with mercury and iodides and made very satisfactory progress. In July there were no organic signs in the nervous system, but the fundus still showed a very slight grade of papillitis. Her visual acuity was then "V. = $\frac{5}{6}$ with either eye." On September 12th I could not detect any trace of swelling, and her vision was "$\frac{5}{6}$ with either eye." The child has been under careful observation ever since, and beyond an occasional headache has been in excellent health. Only the most expert could detect anything amiss with the fundus.

Remarks.—If we are agreed the Wassermann

reaction when undoubtedly positive is conclusive (as it has been in all my cases; I lay no stress on a "weakly positive" reaction), then this child has syphilis. Were we to have another reaction done and it were negative, no stress could be laid upon it. Under efficient treatment the reaction should be negative. A positive reaction would point again to syphilis (especially congenital syphilis) or inadequate treatment. Can we explain the signs and symptoms on any other grounds? I do not think the child has a tubercular focus in one or other cerebellum. The cerebellar signs were never definite. Inco-ordination or paresis of a homolateral limb was never seen. Though uncertain in her walking there was nothing to suggest the cerebellar gait. There was never any tremor or systematised giddiness. Such a low grade of papillitis rapidly subsiding under treatment is not usually seen with cerebellar lesions of whatever type, much less with tubercular lesions. The cerebro-spinal findings admit of tubercle, but the absence of the tubercle bacillus is against it, though it does not, even after special search, absolutely exclude it. The negative von Pirquet is good evidence against a tubercular infection. On the other hand, the findings in the fluid are equally consistent with a mild meningeal infection of syphilitic nature, *e.g.* a trace of albumen and a low grade of lymphocytosis. With regard to serous meningitis, whilst I would admit this as a possibility, I think the symptoms and signs were not sufficiently intense for the picture we usually associate with the acute type of this malady. On the other hand a mild serous meningitis might quite well be due to a specific (syphilitic) inflammation. There is another class of case with which I am especially familiar. It is the type of case we meet with now and again in young women who, in addition to headache, vomiting, and in some cases anæmia, develop signs which are highly suggestive of an intra-cranial neoplasm, viz. bilateral papillitis and paresis of one or both external recti. The condition is not confined to women, so that anæmic thrombosis of the cavernous sinuses or other intracranial veins, as it occurs in chlorosis, cannot be entirely responsible. I have seen eight of these cases in women. Anæmia is not a prominent feature in some cases, as judged by blood-counts, although I am quite certain that iron medication is an absolute specific if the condition is borne in mind as a possibility and iron is given a trial. I have had my patients under observation for years and no further signs of intra-cranial neoplasm have developed. This child, as the blood-count showed, had a good percentage of reds. Unfortunately no record of the hæmoglobin exists. She has got well in spite of iron being withheld. I think we can exclude cerebral abscess with confidence. The course of the case is inconsistent with any such idea. An acute intra-cranial infection of a meningeal type due to some other bacterial invasion can be excluded, in all probability, by the nature of the leucocytosis in the cerebro-spinal fluid. This is by no means true for all cases. I am investigating at the present moment some cases of purulent meningitis occurring in infants in which the cerebro-spinal fluid counts, though given by an expert, were most anomalous, not only in the matter of the cytology but also in the absence of micro-organisms.

(c) A case of acute meningitis occurring in a boy, æt. 11 years, with inherited syphilis is recorded by Billet. During a mild attack of measles nervous symptoms developed suddenly. Lumbar puncture gave a clear fluid with increased tension. No cells or organisms were found. The blood gave a positive Wassermann reaction. The father has suffered from a frontal gumma. Under anti-syphilitic treatment the boy made a rapid recovery.

(d) *General paralysis.*—The meningeal changes which constitute such an important feature in the post-mortem findings in a case of general paralysis of the juvenile type differ in no way from the appearances we find in the adult. They have already been referred to. Pachymeningitis hæmorrhagica occurs in a small percentage of cases of general paralysis in the adult (4·3 per cent., Mott). From the literature on the subject I cannot find whether it is of frequent occurrence in juvenile cases of the inherited type.

From the purely clinical standpoint the meningeal changes in the subject of general paralysis must play a very important part in the picture and symptomatology of the disease, but it would be difficult to attribute any definite symptoms to this particular lesion without calling into play the effects of other lesions which are constantly present.

Hydrocephalus.—A greater or less degree of internal hydrocephalus is a frequent concomitant

of the type of syphilitic meningitis first described above. It may be that some cases of acute serous meningitis (internal hydrocephalus) are due to syphilis. Dr. Mott writes: "Hydrocephalus is extremely rare in acquired syphilis, but has often been observed in the inherited form." He further states that "Elsner has described interstitial inflammation of a specific character affecting the choroidal plexuses" At the present day a careful examination of the cerebro-spinal fluid and the application of the Wassermann reaction should assist us greatly in the diagnosis of hydrocephalus dependent on syphilitic infection. Clinically it differs in no particular respect from hydrocephalus dependent on other causes. It is interesting to note that so shrewd an observer as Henoch "cannot find any connection with congenital syphilis such as is occasionally assumed, nor had he seen the slightest effect from specific treatment in the very few cases in which syphilis could be blamed as a possible ætiological factor."

(6) Hemiplegia, Cerebral Hæmorrhage, Thrombosis, Embolism and Tumour.

Hemiplegia in the young subject, like hemiplegia in the adult, may be associated with a variety of lesions. When of sudden onset polio-encephalitis is a cause, though not a common one (not more than 10 per cent., Batten). The actual cause of this infective disease, as in the case of acute polio-myelitis, is not definitely known. In its usual form syphilis bears no relation to it. Vascular obstruction in an artery, or more especially in a cortical vein (Gowers), is a type of lesion held to be responsible for another variety of infantile hemiplegia. We know that in young adults the subjects of the acquired disease, a hemiplegia with rapid onset is frequently associated with thrombosis in a vessel the seat of endarteritis syphilitica. It would appear from the literature that hemiplegia due to this type of lesion in subjects of the inherited malady is not of frequent occurrence. This to my mind is remarkable, seeing that endarteritis occurs early and is frequent in inherited syphilis. Chiari (quoted by Henoch) described typical endarteritis syphilitica in a child as young as fifteen months. How can the difference be explained? Sir William Gowers, too, states it has been noted in its characteristic form in the subjects of the inherited disease between birth and the tenth year. Dr. Charles Box, in the lecture already referred to, maintains, so far as I can gather, that syphilis may be a factor in the hemiplegias of post-natal type when (1) a hemiplegia develops which has been preceded for some time by premonitory and recurrent convulsions: (2) when separate attacks of paralysis succeed each other at intervals of days or months; (3) when there are prodromal headaches and vomiting; and (4) when an acute hemiplegia develops "silently."

The hemiplegia of young subjects which develops during the acute stage or convalescence from the acute specific fevers need not detain us. It bears no relation to syphilis.

As in the adult with acquired syphilis, so in the young patient the subject of the acquired or the inherited malady, progressive hemiplegia due to a gummatous mass may quite well occur. The records of such cases are few. Byrom Bramwell relates a remarkable case of a gumma of the medulla in a congenital syphilitic boy, æt. 6 years. The family history was very characteristic. The parent developed a tertiary lesion whilst the child was under observation, and this gave a clue to the nature of the lesion in the case of the child. He was admitted "for headache, paralysis of the right arm, right leg, and left side of the tongue. There was a difficulty of speaking and swallowing." During infancy he showed no signs of congenital syphilis so far as could be determined. The paralytic phenomena appeared to date from an attack of measles five months prior to the time the child came under observation. The hemiplegia had developed gradually without any preceding convulsion. "The arm was more paralysed than the leg, and voluntary movements of the paralysed arm were attended by rhythmical jerking movements exactly like the rhythmical tremors which are so characteristic of disseminated sclerosis, but which may also result from pressure on the pyramidal tracts. The tongue was paralysed, atrophied, and the seat of fibrillary twitchings. The paralysis and atrophy were more marked on the left side. The larynx and the pharynx were partly paralysed. There was severe headache, and there had been vomiting. Optic neuritis developed subsequently." The tumour was thought at first to be scrofulous.

As already stated, the development of a typical tertiary lesion in the mother pointed to the correct interpretation of the case in the child, and anti-

specific treatment resulted in considerable improvement in the child's general condition. A careful inquiry into the family history afforded also convincing proof of the syphilitic taint.

Hemiplegia dependent upon *embolism* can only bear a direct relation to syphilis in so far as syphilis may be the cause of the diseased state of the vessel or other site from which the embolus arose. Sir William Gowers relates the case of a boy, æt. 8 years, with inherited syphilis, who died from an extensive *hæmorrhage* into the brain. There was no visible aneurism, and the hæmorrhage had apparently commenced in the right lenticular nucleus, and had burst into the ventricles. The syphilitic disease was mainly confined to the vertebral and cerebellar arteries.

The cranial nerves.—(*a*) *In acquired* syphilis implication of one or more cranial nerves is a very frequent event. The site of the lesion and its character vary in individual cases. In syphilitic basic meningitis the cranial nerves are frequently involved. Squint and ptosis are in a fair percentage of cases the first evidence of a para-syphilitic affection. In gumma of the brain the implication of a cranial nerve may be dependent on increased intracranial pressure, or it may be due to the direct pressure exerted by the tumour mass. In other cases, again, it may be associated with a basal gummatous meningitis. Whilst the paralysis or paresis may be due to a lesion involving the trunk of the nerve, nuclear lesions are sometimes responsible. Acute and chronic forms of ophthalmoplegia are ascribed to syphilis. Optic neuritis, when present, may be associated with the formation of a gumma in any part of the brain, the type and the degree of the papillitis being dependent on the situation of the tumour mass rather than on the fact that the lesion is gummatous. On the other hand the neuritis may be dependent on a basal gummatous lesion, the nerve trunks themselves being involved in the disease. The significance of an Argyll-Robertson pupil is discussed in another paragraph.

(*b*) In *inherited* lues the cranial nerves may be involved in a similar manner. Bilateral deafness of labyrinthine origin appears to be one of the commonest results of the syphilitic poison in these cases. Implication of the third or sixth nerves by a basal meningitis is not an infrequent variety. In rare cases a complete ophthalmoplegia externa is met with in inherited syphilis. Byrom Bramwell records the condition in a little girl, æt. 2½ years. No symptoms of syphilis were noted during infancy, but the patient's elder sister presented a characteristic appearance and the family history was very suggestive. On examination the child was found to be suffering from complete double ptosis, "the lids being mainly raised to the level of the pupils by the action of the occipito-frontales muscles. Absolute loss of the power of raising both eyes, and almost complete loss of the power of depressing them. The internal rectus of the right eye was absolutely and that of the left almost completely paralysed. The eyes stood in the position of absolute divergence. The right eye was used for fixation, but on covering the right the patient was able to fix with the left. The pupils, which measured 4 to 5 mm. in diameter, contracted well to light. There were no ophthalmoscopic signs. The patient looked, and was, markedly stupid and apathetic, in fact, almost idiotic." Prior to the attack she was a bright and active and intelligent child. She was always drowsy and slept continuously. Whilst the patient was under observation she developed typical syphilitic dactylitis. Under appropriate treatment she made a perfect recovery.

(7) General Paralysis of the Insane; Tabes; Diffuse Syphilitic Cerebritis; Pseudo-Tabes; Charcot's Joints and their Significance.

According to Dr. Mott, prior to 1877 general paralysis was believed to be essentially a disease of adult life only. In that year Professor Clouston described a case in a boy, æt. 16 years. He drew attention to the close similarity, both from the clinical and pathological aspect, of the condition as it occurred in the adult and in the young subject. Whilst these parasyphilitic diseases may occur at any age, most of the patients commence to show symptoms at or about puberty according to most observers. At first, in some cases, a fair grade of intelligence exists. In others the children are feeble-minded from the first. Later they become definitely demented. The mental symptoms of the juvenile paralytic resemble for the most part those of the adult. More cases occur in the female sex. In the acquired disease the reverse holds good. Juvenile tabes is much rarer than juvenile general paralysis

(Mott). Whilst the majority of the patients are the subjects of the acquired disease, the smaller proportion is due to syphilis acquired early in infancy or childhood. To establish a diagnosis in doubtful cases the examination of the cerebro-spinal fluid is essential. The more important points have already been referred to in a preceding paragraph.

I have the records of two typical cases of meta-syphilis occurring in young adults. These two patients came under my notice when I was Resident Medical Officer at University College Hospital. They were admitted to the wards under the care of Dr. Risien Russell, and I am indebted to him for permission to place them on record in this connection.

CASE 7.—Albion K—, æt. 20 years, was sent to the hospital for difficulty in walking and defective vision. The father has tabes and the brother has suffered from acquired syphilis. The patient believes he contracted the disease from him. There are no stigmata of the inherited disease. *Examination* reveals the following features in the nervous system: (1) The patient has bilateral primary optic atrophy; (2) Argyll-Robertson pupils; (3) rhombergism; (4) absent knee- and Achilles jerks, and (5) an ataxic gait.

The case affords a good example of adolescent tabes occurring in a young person the subject (in all probability) of the acquired disease. He came under observation prior to the date when lumbar puncture and the Wassermann reaction were carried out as a routine in cases of this nature.

The next case is one of tabo-paralysis.

CASE 8.—Male, æt. 20 years. *History.*—Good health until fourteen years of age when he commenced to develop delusions. His father is a tabetic. *Examination* reveals the following. Although twenty years of age he does not look more than fifteen. He has typical Hutchinsonian incisors. He is dirty in his habits. There is well-marked pes cavus but no other deformity. His gait is distinctly ataxic. His voice is high-pitched, his speech is distinctly tremulous, and there is distinct tremor of the tongue, hands and facial muscles. He suffers from mild though definite grandiose delusions. The pupils are unequal, irregular in outline, and react sluggishly to light. There is no atrophy of the optic discs in this case. The knee-jerks are dull and there is a definite extensor plantar reflex, which is best obtained by stroking the upper outer border of the foot (Barnes's sign).

Here, again, neither lumbar puncture nor an examination of the blood for the Wassermann reaction were performed. This case differs in two main ways from the preceding. Definite grandiose delusions point to implication of the highest centres, and secondly, the patient is the subject of the inherited disease without doubt and not of the acquired. As is frequently the case one of the parents is the subject of tabes. In the former case although the father was also a tabetic it would appear to be an accidental association.

We occasionally meet with cases which at first sight are differentiated with difficulty from instances of true general paralysis. The following is a good example of this type of case, and I am indebted to Dr. Parkinson, my colleague at the Queen's Hospital, for permission to bring the case to your notice. The child was shown at the Children's Section of the Royal Society of Medicine under the title, "A Case resembling General Paralysis of the Insane."

CASE 9.—Susan J—, æt. 8½ years. *History* (Given July, 1911).—Illness commenced four to five months previously with difficulty in walking and marked alteration in the child's disposition. She became bad-tempered and spiteful. She steals things. She vomits occasionally and complains of abdominal pain. As a baby she had slight snuffles.

Family history.—The patient is the fifth child of a family of nine. First, dead infant at seven months. Second, an eight months child, lived three and a half days. Third, living. Fourth, living. Fifth, the patient. Sixth, a miscarriage. Seventh, child died at ten weeks, ? heart disease. Eighth, alive and healthy. Ninth, miscarriage.

Present state.—A dull child, well nourished. Speech is slow and indistinct. There are no stigmata. Pupils are unequal, the left larger than the right. They react sluggishly to both light and accommodation. There is a mild grade of iritis. There is slight ataxy of the lower limbs, but no tremor of hands, lips, or tongue. The left knee-jerk is sluggish and the right variable, but never absent. There is a bilateral extensor plantar reflex. There are no sensory changes. The fundus is normal. The handwriting is good for her age. The *Wassermann reaction* in the blood

was positive. It was repeated several times with the same result.

The cerebro-spinal fluid gave the following: Fluid, clear and no coagulum. Albumen is present, amounts to ·075 per cent. (Aufrecht). Globulin is increased (Noguchi). Glucose decreased (= 0·125 per cent.). Lymphocytes are 40 per c.mm., and there are no polynuclears or organisms. No choline or cholesterol. The Wassermann reaction on two occasions was negative.

The patient was treated with mercurial inunctions and when shown before the Children's Section, (four months later) had much improved. She had gained weight. The pupils were still inactive and unequal. The right knee-jerk is active, and the left is difficult to obtain. The gait is still uncertain, and there is a tendency to scrape the right toes on the ground. Rhombergism, though present before, is not now obtained. Her speech is better, and though her mental processes are slow, she seems more intelligent. The right grasp is a little weak. Except for this there is no paresis. Handwriting good for her age. The Wassermann reaction (blood), as already stated, was positive three times.

In the discussion which followed Dr. Leonard Guthrie said he did not think it was possible to say whether it was a case of general paralysis of the insane or diffuse syphilitic cerebritis. On the whole he was inclined to regard the case as one of diffuse syphilitic cerebritis.

Dr. James Taylor, whilst he hesitated to call the case one of general paralysis of the insane, did not think anyone could say it was not.

To my mind the case is not one of general paralysis, but, as Dr. Guthrie says, one of syphilis of the nervous system. Had there been a positive Wassermann reaction of the cerebro-spinal fluid the diagnosis of tabo-paralysis appears to me to be the more appropriate.

The next case presents some interesting features. When I first saw the child I thought the case might be one of early general paralysis.

CASE 10.—Daisy A—, æt. 7½ years. *History.*—The patient was "a fine child at birth," but commenced to waste at the age of three months. She attended the Children's Hospital, Great Ormond Street, for two months at that time, and beyond this "wasting disease" has had no serious illness. She had no snuffles or rashes at any time. She is very backward, despite the fact the mother considers "she is a sharp child and takes notice of everything." She is a "trying child." Chatters a lot. She is clean in her habits and is not destructive.

Family history.—First, a boy, æt. 12 years, is poorly nourished and has adenoids and a slight ear discharge. Never had any fits or snuffles. No evidence of syphilis. Second, a girl, an eight months child, is dead. She only lived sixteeen days. A very small baby and died with convulsions apparently. The mother was ill during the pregnancy Third, a girl, æt. 9 years, shows no evidence of inherited syphilis. Fourth, the patient. Fifth, a full-term child, a boy. Lived seven months; wasted, but had no convulsions. Sixth, a boy, an eight months child, is now æt. 4½ years. The presentation was a breech one. At one year and ten months had a fit. Since then five other fits. I could find no signs of organic disease of the nervous system and the child presents no evidence of inherited syphilis. Seventh, a girl, æt. 2½ years, is healthy; and eighth, a baby, æt. 9 months, is healthy. Neither show any evidence of syphilis.

Present state (March, 1912).—A well-nourished child. Shape of the head is natural. Rather short for her age. Has a constant and somewhat vacuous smile. No tremor: takes a lively interest in her surroundings and is not at all apathetic. Though seven years old she does not "know her letters" and is still in the "baby class" at an elementary council school. Is very restless and frequently makes grimaces. There is no evidence of chorea. She is clean in her habits; her speech is of the child-like, undeveloped type. There are no enlarged glands. The teeth (incisors) are of the typical "congenital" type and the molars are typical Moon's molars. The bridge of the nose is a little sunken, but beyond these features there are no other stigmata. Heart and lungs are normal. The liver and spleen are not enlarged and the urine is normal.

The Wassermann reaction of the blood is strongly positive.

The nervous system.—Summarised, the main features are as follows:

(1) There is a slight convergent strabismus of the left eye. The left eye (on deviation) is not carried out to the outer canthus as far as normal; the right is.

(2) The pupils are unequal; the right is bigger than the left. The contour of both is irregular. Both respond very sluggishly to light, but well to accommodation. The consensual reactions are sluggish. On shading, the pupils do not dilate as readily as normal. There is no evidence of iritis.

(3) Visual acuity appears to be good, but cannot be estimated as the child does not know her letters or the sounds for them.

(4) Deep and superficial reflexes are normal but there is a distinct tendency for the big right toe to extend. The left is very definitely flexor.

(5) *Fundus.*—The discs are normal but the background is much pigmented (tigroid type). There are no patches of choroiditis and the vessels are good.

(6) There is no tremor, ataxy or spacticity. The gait is normal. The speech is that of a child much younger than her age.

No examination of the cerebro-spinal fluid has been made in this case.

This child has been on mercury and iodides since March, but there is little or no difference in her condition except that the pupillary reaction to light is not so sluggish. It will be interesting to see whether this child in years to come develops any positive sign of syphilis of the nervous system other than the irregular sluggish pupillary reactions.

Within the last few days I have seen another instructive case. He presents features very like the case whose state I have just recorded, and owing to the condition of the right pupil the case merits short description.

CASE 11.—Harry H—, æt. 12 years. He was brought to me because he was very backward, and is only in Standard 2 of an elementary school. He should be in Standard 5 or 6. As a baby he had snuffles and a rash and wasted.

Family history.—The father is a fine healthy-looking man, a labourer. The mother has never had any miscarriages. The first child died when five months old of (?) measles. The second died at one year and four months, (?) cause. The patient is the third child. The fourth child, a boy, æt. 7 years, is healthy. The sixth died at three and a half years from cancrum oris, and the last, the seventh child, is an infant, æt. 16 months, who is stated to be healthy.

Present state.—The boy is very backward. The bridge of his nose is depressed, the incisors (upper) are notched. He is moderately well nourished. His aspect is that of a subject of hereditary syphilis. He is not deaf.

The *nervous system* is normal except for the pupils. These are equal, but the reaction of the right to light is very sluggish. The contrast in this respect with the other, the left, is very striking. There are no iritic adhesions, and the media are normal. The fundi are deeply pigmented, but there are no patches of choroiditis. The optic nerves are a little paler than normal, and there is some undue pigmentary disturbance in their immediate neighbourhood. The visual acuity is V. $= \frac{6}{9}$, either eye.

In the case of this patient again I have had no opportunity to examine the blood or the cerebro-spinal fluid. In my own mind I have little doubt the child is suffering from inherited syphilis, which is responsible for the low grade of mentality and the state of the right pupil.

An Argyll-Robertson pupil of the classical type is strong evidence of syphilis (Babinski, Mott and others). According to most observers its presence is suggestive rather of parasyphilis than syphilis. Unilateral pupil phenomena of this type are uncommon. Iritic adhesions of course must be excluded. Under mercurial treatment a sluggish reaction may become active again. It must be borne in mind the absence of the light reflex is also a valuable sign of disease of the third ventricle and the structures in its immediate neighbourhood, *e.g.* cysts of the ventricle and lesions of the thalamus (Buzzard).

The presence of a *Charcot's joint* in a young subject is highly suggestive of syphilis. I have only seen one such case.

CASE 12.—*The patient was a boy, æt. 16 years.*—He sought advice for swelling of the right knee-joint and difficulty with his walking. He looked very delicate. The knee-joint was much swollen, and partial subluxation of the joint was present. It presented the features typical of the hypertrophic type of the malady: subluxation, soft crepitis, hypertrophied synovial folds with loss of the normal outline. The right ankle-joint presented a very similar appearance. The affection was quite painless. The knee-jerks were absent, the pupils were sluggish in their reaction to light, and there was distinct blunting to pin-point on the lower limbs up to the knees. There were no signs of syringomyelia: no nystagmus, atrophic paralysis, and deformities other than those already mentioned. No dissociated anæsthesia and no history of painless whitlows or other characteristic features could be determined.

The occurrence of a Chatcot's joint in a young subject under twenty years of age must be exceptional (Barbier and Gassier). The syphilitic epiphysitis of infants and the dactylitis of older children could hardly be confounded with this lesion. We are all familiar with the symmetrical synovitis of the knees which occurs in young subjects at the age when interstitial keratitis is so liable to be found.

Spontaneous fractures or fractures associated with slight, and at other times inadequate injury, occur in adults, the victims of tabes. Dr. Mott relates the case of a young man suffering from tabo-paralysis (presumably the subject of the acquired disease), who susatined a fracture of the thigh, which proved fatal. The spinal disease was not discovered during life, but microscopic examination of the cord showed the characteristic changes. The subject has been dealt with at length by A. Guichard. His thesis contains the histories of sixteen cases.

(*To be concluded.*)

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A CLINICAL LECTURE

ON

TREATMENT OF HÆMOPTYSIS.

By OTTO GRUNBAUM, M.D.Cantab., F.R.C.P.,
Assistant Physician to London Hospital.

Gentlemen,—I have chosen the treatment of hæmoptysis as a subject to talk to you about to-day, because it is one which we all have to deal with at comparatively frequent intervals.

It so happens to have been the first condition which I was called upon to treat after I had obtained medical qualifications.

On looking back upon that case I see that there was much to be learnt from it, and when I relate to you some of the details you will agree with me that it epitomises a great deal of medicine.

Hæmoptysis occurs from several causes, and it is absolutely necessary that the cause shall be determined before any treatment is adopted.

We shall see shortly that when hæmoptysis is due to infarction of the lung, the treatment indicated is almost the contrary to that which should be adopted when hæmoptysis is due to destruction of an artery through infection with any micro-organism or new growth.

Some time ago a friend of mine was spending his holiday in the Hebrides, and whilst fishing he had to lean over the side of the boat; he felt something warm and salt in his mouth; on spitting he saw blood on the surface of the sea. Believing that he was suffering from hæmoptysis he called to some friends in another boat to come to his assistance. Instead of complying with the appeal they asked him "Why?" and on his saying that he must have broken a blood-vessel in his lungs, they roared with laughter at him. The explanation was that they could see that blood was trickling into his mouth from his chin, which he had cut whilst shaving that morning, and had started bleeding

owing to the stretching of the skin on his leaning over the side of the boat.

Hæmoptysis may not be due to anything as obvious as this, but we must search the mouth, the nose and the throat before we should come to the conclusion that it is due to anything happening in the lungs and trachea.

It is quite remarkable how frequently the history of hæmoptysis leads the doctor to find a lesion in the lungs. I have seen quite a number of people in whom hæmoptysis has been asserted to have been due to tuberculosis, when it has really been due to some small vein giving way in the throat, the lungs being perfectly normal.

Not infrequently a high blood-pressure assists one to arrive at the correct conclusion.

Having satisfied ourselves that the blood comes from the lungs, there are three common causes. The commonest is one that is not often recognised, namely, emphysema.

Nearly 50 per cent. of cases in whom emphysema has developed, some time or other have expectorated streaks of blood with the sputum. This happens in London, whilst it does not happen in the country or in the smaller towns.

One can easily understand that destruction of the septa between the air-spaces leads to the severance of small arterioles, and these bleed and lead to hæmoptysis.

The other two causes are tuberculosis and mitral stenosis.

There are two forms of hæmoptysis in mitral stenosis. One is due to congestion and oozing from the capillaries, and the other due to hæmorrhage following infarction.

The form due to congestion may be treated with contempt.

The latter is a condition in which the treatment must be almost the direct opposite to that which would be adopted were the hæmorrhage due to ulceration of blood-vessels.

Having decided that the hæmorrhage is not due to any cardiac condition, and in all probability is due to tuberculosis, the treatment should consist of rest. Rest may be divided under the headings of mental rest, physical rest, and physiological rest.

We cannot obtain complete rest in any of these ways for any considerable length of time, but we can do our best to approach the ideal.

Mental rest should be attempted by assuring the patient and his friends that hæmoptysis is not a serious condition, and that it will stop and the patient will be no worse after than before, and in addition to this the patient may be given a small injection of morphine provided that the hæmorrhage or secretion from the lungs is not so profuse as to lead to asphyxia were he not to cough for some hours.

The next point is physical rest.

The patient should be placed in bed and not allowed to move for any reason. If the side of the hæmorrhage is known, then the patient should be laid on the affected side so as to diminish the risk of the blood flowing into the air-tubes of the healthy side.

If the hæmorrhage has been very profuse it might be wise for the patient to lay quite flat without a pillow. I think the presence or absence of a pillow should be determined purely by the inclination of the patient and not arbitrarily by the medical man or nurse. Some patients can lie flat with comfort, whilst others who are accustomed to sleep with many pillows may be totally unable to sleep when lying flat.

When it is difficult to make the patient stop still an ice-bag may be applied to the chest, and instruction given that it is important that it does not shift.

Personally I do not think that an ice-bag as a detractor of heat is of any therapeutic value. In cases of this kind it may assist in making the restless patient control himself.

If hæmorrhage continues in spite of treatment, which we shall refer to shortly, then enforcement of complete physical rest by the application of strapping to the affected side should be considered, but this is a treatment which should not be adopted as a routine.

I have often thought about the wisdom of giving inhalation of oxygen in order that the chest movements may be reduced to a minimum, but if oxygen is administered it must be done without exciting the patient or interfering with him in any way.

One must remember that the method which may be the most efficient in theory is not necessarily the best in practice. Allow me to explain what I mean by this somewhat extraordinary statement.

Some years ago experiments on the administration of oxygen were being carried out at the London Hospital. A face-piece of celluloid surrounded by flexible material was applied to the

patient's face ; this allowed almost pure oxygen to be inhaled.

The first patient on whom it was tried was suffering from heart trouble and was gasping for breath. On having the face-piece applied he had the sensation of oppression, and of being unable to breathe, and this in spite of a plentiful supply of oxygen, and said that he would willingly pay another patient for it to be tried on him.

In this case a method which had many theoretical advantages did not succeed in practice.

Possibly the simplest method of supplying oxygen is to obtain a rubber teat, cut a fair size hole in it and attach it to a flexible rubber tube, taking care, however, that the oxygen shall pass through a metal tube immersed in hot water or bubble through hot water so that it shall be warmed before it reaches the patient. The rubber teat is inserted into the nostril and it does not inconvenience the patient in any way.

This assists in the production of physical rest.

Next is the question of physiological rest.

This is naturally impossible, but an attempt should be made to diminish physiological activity, and especially that of the circulatory system—by making the heart beat as slowly as possible, and reducing the blood-pressure in the pulmonary system to a low pressure.

The first thing which should be done is to give a mild aperient. This produces an effect in at least three ways.

By making the bowels act it prevents the accumulation and absorption of products of decomposition in the intestine of the tyramine series, which we know tend to raise blood-pressure.

Secondly, the diminution in the absorption of fluid prevents hydræmic plethora.

Thirdly, the irritation of the intestinal wall leads to dilatation of the blood-vessels, and thereby diminishes the quantity of blood in the rest of the circulatory system. In my opinion it is the first of these which is of the most importance.

One must remember that a powerful purge is contra-indicated because the patient must not be violently disturbed, and strain of any kind must be avoided.

The next question which arises is, Do any drugs assist in slowing the heart and lowering the blood-pressure, especially the blood-pressure of the pulmonary system ?

Whenever we use a drug we must satisfy ourselves that the beneficial effect produced by it is greater than any possible detrimental effect.

I should like to impress upon you, gentlemen, that occasionally symptomatic treatment varies very far from curative treatment. In cases of hæmatemesis and of hæmoptysis symptomatic treatment may be responsible for the death of the patient. A patient may feel faint owing to low blood-pressure ; raising the blood-pressure by injecting strychnine may remove the faintness, but it is quite possible that the rise of blood-pressure will lead to fresh hæmorrhage and lead to death.

In the past ergot was the routine treatment for hæmoptysis, and I cannot help thinking that it was introduced by false analogy.

It was found that an injection of ergot led to the cessation of hæmorrhage from the uterus, and it was thought that if a drug prevented hæmorrhage from the uterus it would prevent hæmorrhage from the lungs.

I told you earlier that the first patient which I was called upon to see, after I had become qualified, was suffering from hæmoptysis.

He was a man who happened to be the managing director of a famous danseuse. When this lady heard that her managing director was suffering from hæmorrhage from the lungs she immediately sent him a Chapman's bag.

A Chapman's bag consists of a long india-rubber bag, which Chapman used to have filled with ice and applied to the spinal columns of his patients, and with it believed that he cured many diseases.

This danseuse wrote that for some years she had been able to dance continuously by applying the Chapman's bag to her back when the catamenia were due, and she felt certain that since this treatment prevented hæmorrhage in her case it would be successful in that of her friend.

The letter struck one as humorous, but really it is no more humorous than the treatment of hæmorrhage by an injection of ergot, the only difference being that in one case it has been advised by qualified medicos and in the other case by a danseuse.

The great objection to ergot is that it raises the blood-pressure, but it is within the limits of possibility that a dose will lead to fatigue of the myoneural junction after it has been stimulated by ergotinine administered either subcutaneously or

intra-venously, and thus the blood-pressure will become a shade lower than it would be without the administration of the drug. So that although ergot may do harm, it is within the limits of possibility that it may do good. This to my mind does not justify its routine use.

Another drug which has also been abused is adrenalin. Adrenalin given by the mouth is harmless because it does not raise the blood-pressure. Given intra-venously or subcutaneously it raises blood-pressure for a time, but it may fall to a level lower than the normal for a short time afterwards.

Years ago, an American, whose name I will not quote because I do not wish to be involved in any libel action, asserted that fourteen consecutive cases of hæmoptysis were benefited immediately by the injection of supra-renal extract. It was before the days of the isolation of adrenalin. This experience is different to that of most other observers.

One has got to be extremely careful in placing reliance on observations recorded in some American journals, although there are a few excellent journals which are conducted as well or better than any of the European.

To illustrate this I might relate to you an instance told to me by a colleague who was collecting cases of a rare disease, and who found three or four recorded in an obscure American journal. The editor put him into communication with the author of the paper, who wrote that he advised my friend not to take too much notice of his article because he had written it some years ago when he was on holiday and had nothing else to do. In other words he admitted that the cases did not really exist, but had been built up by his imagination in order to make an interesting paper.

I do not suggest that the fourteen consecutive cases of hæmoptysis which were beneficially treated by supra-renal extract were all imaginary, but I do suggest that the improvements were to a great extent imaginary.

In my opinion aconite is the most useful drug, because it slows the heart by stimulating the vagus centre, and also by its depressing effect upon the heart muscle leads to a fall in blood-pressure.

In the administration of aconite it is my custom to prescribe two minims of the tincture every hour; this may be continued until the pulse becomes abnormally slow, that is, below 65 per minute, or irregular.

If possible the blood-pressure should be measured: a fall below 90 mm. Hg. is an indication for stopping the aconite.

Further treatment consists of increasing the coagulability of the blood.

This may be attempted by the administration of calcium, preferably as calcium lactate; 30 gr. may be given three times a day.

Calcium lactate is soluble in boiling water, and is not especially unpalatable when given in solution, but it may be almost impossible to give a powder even as a cachet because the production of unnecessary coughing must be avoided.

The next important question is food.

It always seems to me that the medical profession does not fully realise that a man can live quite a long time without taking food, and often no harm will result from the patient being supplied with fluid only for several days.

The patient will of course feel weak, and the relatives will have to be assured that the absence of food is absolutely necessary, because there is a tendency to believe that the patient must be fed at all costs.

Occasionally, in spite of all the treatment I have described, hæmorrhage continues.

What else may be done?

The case I have referred to several times already was at an altitude of 3500 feet when the hæmoptysis began, and the hæmorrhage continued in spite of all treatment for quite a number of weeks until the patient was moved to a lower level, and then it ceased.

In all probability an obstinate case of hæmoptysis should be moved to as near the sea level as possible.

The side should be strapped in order to diminish the movements to a minimum, and if, in spite of this, hæmorrhage continues, one must consider seriously the advisability of producing pneumothorax, but of course this can only be carried out if there is not the slightest doubt as to which side is giving rise to the hæmorrhage, because pneumothorax on the wrong side would increase the movements of the affected side and so diminish the chance of the recovery of the patient.

If all these things are without avail I own to being at the end of my resources, but there are treatments which have been suggested, and I should like to mention these to you and offer criticism.

Placing bandages round the limbs in order to compress the veins will increase the amount of fluid in the limbs and thereby diminish the quantity of blood in the pulmonary circulation, but this cannot be maintained for any length of time; it may do good for a short time, but I do not place much reliance in it.

Another question is the possibility of bringing a styptic to the bleeding-point, and thereby causing contraction of the ulcerated vessels.

It is theoretically possible to atomise a solution such as that of adrenalin and inhale it, by this method bringing it near the bleeding-point, but the amount it is possible to introduce in this way is very small, and the blood coming out of the vessels would undoubtedly wash the traces of it away and prevent the vaso-constrictor reaching the plain muscle of the artery.

However, as long as a comparatively harmless fluid, such as a solution of adrenalin, is used there is no great objection to the method being attempted.

Some bolder spirits might wish to try perchloride of iron or turpentine or some irritating substance of this type, which would lead to coughing and much more damage than possible good.

Experience teaches one that due consideration is not always paid to the pharmacological properties of a substance before a new method of administration is carried out.

Some years ago a physician said to me that he regretted he could not carry out intra-tracheal injections because he was anxious to inject ipecacuanha in cases of bronchitis. He thought that if he could get the drug to the seat of the disease much greater beneficial effects would result than when it was given by the mouth.

Well, gentlemen, I cannot help thinking that it was fortunate that this man could not undertake intra-tracheal injections, because, as you know, ipecacuanha is used to a large extent as an expectorant because it stimulates the stomach, and since it is a very violent irritant the introduction of it into the trachea would have led to irreparable damage.

Personally, I am against intra-tracheal injections in any disease, but in hæmoptysis I fail to understand how the drug can reach the part affected, and therefore I think it is contra-indicated.

Another form of hæmoptysis, namely, that due to infarction of the lung, which is usually accompanied by stenosis of the mitral valve, requires treatment the direct opposite to that of hæmoptysis due to ulceration of the blood-vessels.

One cannot deal with an infarction which has actually taken place, but one can direct treatment to prevent the recurrence. It is true that the treatment will not always be successful.

Hæmoptysis of this type is produced by a small clot being carried to the lungs.

It is remarkable at first sight that a disease of the left side of the heart should lead to clots forming on the right side. It is true there is an alternative theory, that the clot forms on the left side of the heart and is carried to the lungs through the intercostal vessels, which also supply part of the lung tissue, but this is a theory which is not generally accepted.

Stenosis of the mitral orifice leads to a slower circulation. The circulation through the ventricles is so tardy as to allow clots forming behind the columnæ carneæ, and this is carried through the pulmonary artery to the lungs.

When an infarction of the lung occurs the patient is suddenly breathless and sits up in bed; the heart beats rapidly; localised pain similar to that occuring in pleurisy may be complained of.

This shows the absurdity of calling an attack pulmonary "apoplexy."

You all know that apoplexy is derived from ἀποπλήσσω, which means "I strike down."

When an infarction of the lung takes place the patient has to sit up. It is true that there is hæmorrhage in the lung following the embolus, just as occurs in the brain in cases of apoplexy.

The quantity of blood expectorated in pulmonary infarction varies tremendously according to the position and size of the embolus. Gradually compensation is restored, a piece of lung has been thrown out of action, but with this loss the other part of the lung can carry on the work.

The next thing to be done is to prevent the recurrence. This can be brought about by stimulating the circulation and also by diminishing the coagulability of the blood, and it is chiefly in the latter direction that benefit can be obtained.

The coagulability of the blood may be diminished by administering sodium citrate. A drachm of this may with advantage be added to every half pint of milk. Calcium citrate is precipitated and absorbed with difficulty; this leads to the diminution of the coagulability of the blood.

I remember on one occasion in the London Hospital the patient had had pulmonary emboli at intervals of about three weeks for several months. She was put upon a drachm of sodium citrate four times a day. She knew it was added to the milk, but she did not know the object of having it added, and was under the impression that it was an hypnotic. She used to sleep for one and a half hours after every glass of milk containing the citrate.

When I told her that sodium citrate was not a hypnotic she refused to believe me and said she knew better. She benefited considerably by this addition and had no embolus for six months. She finally died, but not from pulmonary embolus.

As regards improving the general circulation I fear that time does not permit of my going into details on this matter; it is rather a lecture on the treatment of cardiac disease and hæmoptysis.

November 4th, 1912.

LECTURES
ON
DISEASES OF THE PLEURA.*

By CECIL WALL, M.D., F.R.C.P.,
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LECTURE III.

Serous Pleural Effusions and their Treatment.

TO-DAY I purpose considering the indications for treatment of pleural effusions. It is obvious that a general view of the ætiological factors and concomitant conditions must be taken before the adoption of any active measures. I have already considered some important points in connection with the treatment of simple dry pleurisy, and to-day I will begin with some observations upon the nature of serous pleural effusions, and the indications for treatment that may be derived from their study.

Unfortunately we still remain in almost complete ignorance with regard to the mechanism of production and absorption of pleural effusions. It is said that when the pleura is inflamed the stomata become blocked so that secreted serum is not absorbed.

I am told, however, that stomata have not yet been demonstrated in the human pleura. I know that effusions are found in the post-mortem room quite commonly with evidence of very small areas of pleural inflammation, and very extensive pleurisy with very slight effusion, and I have read of Starling and Tubby's experiments by which they showed that methylene-blue and indigo carmine injected into the pleural cavity appears in the urine within five minutes but does not reach the thoracic duct for an hour or more.

Consequently the theory which ascribes a pleural effusion to the blocking of the stomata seems no longer tenable, and any treatment based upon it requires cautious investigation. In practising the art of medicine we are necessarily very much influenced by theory, and we ought continually to examine and test the premises upon which the theories are based. This is my excuse for drawing your attention to the weakness of a received explanation of pleural effusion without being able to suggest a more satisfactory rival. No useful purpose would be served by the discussion here of the parts played by filtration and osmosis in the secretion and absorption of fluid into and out of a serous sac.

The first point which we have to decide in any given case is whether the effusion is passive, *i. e.* due to physical causes, or active, *i. e.* due to inflammation.

Passive effusions are sometimes described as transudates, those caused by inflammatory processes are termed exudates.

Passive effusions or transudates into the pleural cavity produce the condition known as hydrothorax or dropsy of the pleura.

Hydrothorax is met with most commonly in renal or cardiac disease; in renal disease it is probably the result of the presence in the blood of certain toxic products, and is usually, though not invariably, associated with œdema elsewhere. As might be expected, renal hydrothorax is usually bilateral, thought it is common to find that there is more effusion on one side than the other.

In cardiac disease hydrothorax probably is determined by vascular disturbance and is frequently unilateral, the right side being more often affected than the left. The small passive effusions so often found in the post-mortem room which apparently developed during the terminal phase of life probably have a similar mechanical cause.

Hydrothorax has been found in association with malignant disease of the lung or mediastinum, thoracic aneurysms, cirrhosis of the liver and beriberi, and I will show you a case in which the presumptive evidence is in favour of the effusion being due to angio-neurotic œdema. We suspect that an effusion is a transudate if it is found in association with one of these conditions; confirmation of the suspicion may be obtained by examination of the fluid chemically, microscopically and bacteriologically. Exudates are of inflammatory origin; examination of the fluid may aid in their differentiation from transudates and give a clue to the nature of the causative infection.

Chemical examination of pleural effusions shows that they may differ markedly in composition.

To-day I will consider those effusions which are serous and sero-fibrinous, and will leave for subsequent discussion those which are hæmorrhagic or purulent and contain fat (chyliform), bile-pigment, cholesterin and other peculiar constituents.

* Delivered at the London Hospital.

Speaking generally transudates are found to have a low specific gravity (1·015 or under) with only small quantities of albumen (less than 3 per cent.). This albumen is said to be chiefly serum albumen or serum globulin with a trace of fibrinogen; as a rule no clot forms unless blood be added.

Exudates have a specific gravity of 1·018 or higher and contain at least 4 per cent. of albumen; they contain more fibrinogen, and usually form a clot without the addition of blood or fibrin ferment. In addition to serum albumen and serum globulin they contain a mucleo-albumen and a mucoid substance. The presence of the nucleo-albumen (euglobulin) is sometimes taken as a differential test between the two classes of fluid; a few drops of acetic acid added to an exudate will cause a cloudy precipitate of euglobulin. In transudates this cloud is very faint.

Dr. Tidy has analysed for me the effusion in two cases which I will show to you at the end of the lecture.

The first case is that of a pleural effusion in association with chronic nephritis, and partakes of the nature of a transudate: Specific gravity, 1·012; reaction alkaline; albumen coagulated on boiling, 0·17 per cent; globulin, a trace; mucin, absent; urea, a trace; lecithin, absent; total solids, 1·26 per cent., *i. e.* organic 0·49 per cent., inorganic 0·77 per cent.; including chlorides, 0·54 per cent.; phosphates, 0·13 per cent.; depression of the freezing-point, — ·64° C.

The second specimen was taken from a patient suffering from a tuberculous hydro-pneumothorax: Specific gravity, 1·022; reaction alkaline; albumen coagulated on boiling, 0·95 per cent.; globulin, 1·07 per cent.; mucin, a large quantity; urea, a trace; lecithin, absent; total solids, 6·67 per cent., *i. e.* organic 6·07 per cent., inorganic 0·60 per cent.; including chlorides, 0·34 per cent.; phosphates, 0·018 per cent.; depression of the freezing-point, — ·47° C.

Microscopic examination is concerned chiefly with the determination of the cellular elements of the effusion.

Red blood-corpuscles may be found, but are not of great importance in diagnosis because so many different causes may lead to their presence.

The white cells are classified as follows:

A. Polynuclear: (*a*) Neutrophiles (finely granular oxyphiles), (*b*) eosinophiles, (*c*) mast-cells. B. Mononuclear: (*a*) lymphocytes, (*b*) endothelial cells, (*c*) cells intermediate between (*a*) and (*b*).

These cells often show degenerative processes, and their recognition is not quite so easy as in examination of the blood.

Eosinophiles and mast-cells are not very commonly found. The endothelial cells are large, flat, irregular, round or oval in contour, with a round or oval nucleus which is often vacuolated. They may be isolated or in groups, suggesting their origin from the pleural surface. They are phagocytic.

Widal claimed that a predominance of polynuclear cells suggests an infection by a pyogenic organism (pneumococcus, streptococcus or staphylococcus), lymphocytes, by the tubercle bacillus and endothelial cells an effusion of mechanical origin. Further investigation has shown that such a claim cannot be universally substantiated; it is now held that the character of the cells depends not only on the cause of the process but also on the activity of the pleural inflammation.

The presence of many polynuclear cells suggests strongly a pyogenic infection, but lymphocytosis may occur in conditions other than tuberculosis, for instance in sarcoma; in some cases of tuberculous effusion, especially in the earlier stages, the predominant cells may be polynuclear. Dr. Inman gives the following examples:

(1) A case of chronic pulmonary tuberculosis with a pleural effusion (tubercle bacilli in the sputum): Total leucocytes per c.mm., 9375; small lymphocytes, 12 per cent.; large lymphocytes 5 per cent.; neutrophiles, 75 per cent.; endothelial cells, 8 per cent.

(2) A case of chronic pulmonary tuberculosis with a pleural effusion (tubercle bacilli in the sputum): Total leucocytes per c.mm., 13,750; small lymphocytes, 88 per cent.; large lymphocytes, 5·5 per cent.; neutrophiles, 3·5 per cent.; endothelial cells, 3 per cent.

(3) A case of acute tuberculous pleurisy: Total leucocytes per c.mm., 10,625; small lymphocytes, 75 per cent.; large lymphocytes, 14 per cent.; neutrophiles, 2·5 per cent.; endothelial cells, 7·5 per cent.

(4) A case of chronic renal disease with hydro thorax: Small lymphocytes, 24 per cent.; neutrophiles, 13 per cent.; endothelial cells, 63 per cent *N.B.*—This fluid was removed in the post-mortem room.

(5) A case of mediastinal lympho-sarcoma with a pleural effusion: Total leucocytes per c.mm., 6000; small lymphocytes, 58 per cent.; large lymphocytes, 8 per cent.; neutrophiles, 18 per cent.; endothelial cells, 14 per cent.

(6) A case of mediastinal sarcoma with a pleural effusion: Small lymphocytes, 95 per cent.; neutrophiles, 3 per cent.; endothelial cells, 2 per cent.

(7) A case of mediastinal sarcoma with septic broncho-pneumonia and a pleural effusion: The specimen was obtained at the autopsy fourteen hours after death. Small lymphocytes, 20 per cent.; neutrophiles, 75 per cent.; endothelial cells, 5 per cent.

These figures are sufficient to show that though a cytological examination of a pleural exudate may give valuable confirmatory evidence in the diagnosis, yet it cannot be considered sufficient to establish the diagnosis.

Occasionally cancer-cells and sarcomatous spindle-cells may be found in pleural exudates. As a rule, however, in malignant disease the cells are chiefly endothelial and small lymphocytes.

Bacteriological examination is often of the greatest value in determining the ætiology of a pleural exudate. Cultivation may demonstrate the presence of pneumococci or of pyogenic streptococci or staphylococci.

If no growth is obtained on cultivation it is usually assumed that the effusion is either tuberculous or a passive transudate.

Microscopic examination of a smear preparation may reveal the presence of some organisms. Pneumococci, streptococci, and staphylococci may thus be recognised. Pneumococci are usually extracellular, but occasionally they have undergone phagocytosis.

Dr. Tidy has kindly lent me some specimens which demonstrate these points.

The discovery of the tubercle bacillus in a pleural effusion is not easy.

Jousset, in 1903, suggested an ingenious method which goes by the name of "inoscopy." The fluid is allowed to clot. The clot is then digested with pepsin and hydrochloric acid; the digested fluid is centrifugalised and the deposit examined by the Ziehl-Neelsen method.

Emerson remarks, "Jousset obtained astonishingly good results, so good that his critics hint that he mistook masses of hæmoglobin or fuchsin and scratches on the glass, etc., for tubercle bacilli."

Others, like Emerson, have failed to get satisfactory results from inoscopy.

Other methods have been tried for the discovery of tubercle bacilli in pleural effusions, and different observers have claimed success in from 22 to 85 per cent. Jousset claimed 100 per cent. of successes for his method. From the frequency with which pleural effusion is found in association with miliary tubercles in the subpleural lymphatics spreading from the bronchial glands, I am inclined to suppose that though the effusion may have a tuberculous origin, yet free tubercle bacilli may not be present in it. The test of inoculating a guinea-pig with some of the effusion is perhaps the most satisfactory, though sometimes the result is negative, even though the subsequent history demonstrates the case to have been tuberculous. The method is only possible in certain circumstances, and at the least requires an interval of a month or six weeks before the result can be learnt.

I have tried it in two cases. The first was in a man who had an attack of pleurisy with effusion in 1903. The cells in the fluid were all mononuclear; no organisms were seen on microscopic examination. Some of the fluid was inoculated into a guinea-pig, which was killed a month later; generalised tuberculosis of the mesenteric glands was found. The patient recovered from the pleurisy and has remained in good health since then.

The second case was in a girl, æt. 10 years, who developed a pleural effusion in 1905. Clear yellow fluid which clotted on standing was withdrawn with an exploring needle; this contained many mononuclear cells but no visible organisms; cultures remained sterile.

A guinea-pig was inoculated with 3 c.c. of the fluid and killed after twenty-six days; in the interval it had gained weight (255 grm. to 429 grm.), and on examination all the organs were found to be perfectly healthy. Since then the patient has remained perfectly well.

The observations in both these cases were made for me by Dr. Bulloch.

Lord obtained fifteen positive results out of sixty-six cases (22·7 per cent.). Eichorst injected 15 c.c. and obtained evidence of tuberculosis in 62 per cent. Le Damany injected much larger quantities, 50 c.c. up to 300 c.c. in repeated doses, and showed the tuberculous nature of forty-seven out of fifty-five cases of pleural effusion.

When the diagnosis has been completed the problem of treatment must be considered. If the effusion is passive and associated with increasing dyspnœa, cyanosis or sense of oppression, prompt removal is indicated. Kingston Fowler remarks very truly that "next to an unsuspected tuberculosis an enormous hydrothorax is the most common revelation of the post-mortem room," and urges the

importance of examining the chest of every patient however urgent the symptoms pointing out the relief which so commonly follows the aspiration of a passive pleural effusion.

The operation is devoid of risk if the rules of asepsis be observed; it must be remembered that in renal disease there is a very great liability to septic infection.

With regard to exudates Lord remarks that "tuberculosis occupies the first place in the ætiology of pleural effusions, but all sero-fibrinous effusions cannot be classed as tuberculous." In my experience exudates which are not tuberculous either are absorbed rapidly without interference or more frequently require treatment by resection of a rib and free drainage.

Experience seems to show that even if the first specimen of an effusion is found to be sero-fibrinous, but contains polymorphonuclear cells and pyogenic organisms, that sooner or later it will become an empyema and require treatment as such.

In discussing the treatment of empyema I shall have occasion to point out the disadvantages of any less radical measures than resection of the rib and free drainage.

At present we will confine our attention to serous or sero-fibrinous effusions which are presumably of a tuberculous nature.

During the acute stage while there is fever it is obvious that the patient should be confined to bed and measures be adopted for the relief of symptoms.

It seems to me extremely doubtful whether any measures such as the limitation of fluids in the diet, the administration of diuretics or the local application of hygroscopic substances such as compresses strong of salt solution or antiphlogistine have any influence on the absorption of a pleural effusion. Blisters should be avoided; they are of doubtful utility, and increase the risk of sepsis should aspiration become necessary. Poultices or hot-water bottles usually suffice for the relief of pain.

I assume that to complete the ætiological diagnosis a specimen of the fluid has been obtained early and submitted to examination. The removal of a small quantity in this way cannot have any therapeutic effect. Aspiration of larger quantities of fluid as a measure of treatment requires most careful consideration. All are agreed that in certain circumstances removal of at least some of the effusion is necessary. If the effusion is so large that it obviously causes mechanical embarrassment of the circulation and respiration immediate relief is demanded. The development of bronchitic *râles* in the opposite lung is usually taken as a sign that delay would be dangerous. If after a lapse of two or three weeks the effusion does not seem to lessen in amount all agree that some should be removed. There is, however, a divergence of opinion upon the advisability of removing in any case all the fluid that will come away on aspiration, and still greater divergence upon the advisability of early aspiration. The arguments for and against early tapping and complete as opposed to partial removal of the fluid are similar, and may be discussed at the same time.

In favour of delay is the fact that medium and small effusions are often spontaneously absorbed, and the patient is thereby saved the discomfort of the operation and the risks that may attach thereto.

The case against delay is urged eloquently by Dr. Philip in a lecture published by him in the 'Medical Press and Circular' of January of this year:

"Are we to tap? If so, when? It is often said, 'Wait and see, judge by results. Tap, if in spite of treatment, fluid is collecting, or tap if symptoms, especially dyspnœa, be present in excess.' My answer is, Tap, and tap early. Why wait until the lung is thrown into a state of functional disability? Why allow collapse, and run the risk of adhesions of imperfect expansion afterwards? Why not remove the fluid which is the resultant of a flaw in the lymphatic circulation? Why not save the inconvenience to the heart and other organs which in many cases supervenes? Why not get rid of the toxin present in the fluid? Yes, tap, and tap early, believing that you thereby anticipate symptoms, lessen the chances of permanent disability of the lung and limit present circulatory lung embarrassment.

"Do not be unduly scared by the bogey of disadvantages which may follow early aspiration. So far I have failed to meet them. It used to be said that the fluid in the pleural sac acted as a splint to the tender and diseased lung. This is poetry rather than sound theory. To try to realise it in practice is fallacious. It has also been suggested that in tapping you are liable to produce pneumothorax. So far in frequent tappings I have not had this occur. It certainly cannot be a common incident. Should you be unfortunate enough to induce it, what then? In place of a cushion of fluid you have a cushion of air, and no serious consequences are likely to ensue.

"You are sometimes warned that a patient may die during aspiration. This is, fortunately, of rare occurrence. In judging of the possibility you must keep in view that the patient with pleural effusion may die suddenly apart from aspiration."

These are strong words from a physician in authority; they are not backed by figures, but are favoured by some statistics published by Traley in the 'American Journal of Medical Sciences' for May, 1907, and quoted by F. T. Lord. Of fifteen uncomplicated cases treated by tapping, the average duration of effusions occupying one third of the pleural cavity was 15·4 days, while in seventeen cases

of the same degree not tapped it was 21·8 days. Thirty-nine cases with effusions occupying two thirds of the pleural cavity treated by tapping lasted 20·1 days, whereas fifteen cases of similar severity left alone lasted 24·3 days. These reasons urged by Dr. Philip require most careful consideration. He asks why we should wait until the lung is thrown into a state of functional disability and run the risk of subsequent imperfect expansion. He pours scorn on the suggestion that the fluid acts as a splint to the tender and diseased lung. It may be urged in reply that the risk of subsequent imperfect expansion is admitted to be very slight, and that a delay of a fortnight or three weeks is unlikely to increase the risk. It would appear that Dr. Philip assumes for the purpose of this argument, that a tuberculous effusion cannot have any beneficial effect upon the primary focus of disease, that it is a reservoir of toxins and wholly disadvantageous. It always seems to me to be wise to assume that natural processes have some teleological significance unless we can prove otherwise. Dr. Philip would, I think, admit that the best treatment for an active tuberculous gland would be to save it from physical disturbance. He quotes a case in support of the view that I have already urged that a pleural effusion frequently owes its origin to a tuberculous bronchial gland, and yet he thinks that Nature's method of securing physical rest to the inflamed gland by permitting collapse of the lung should be prevented.

I have seen two cases of pleural effusion in which tapping was followed about three weeks later by acute general miliary tuberculosis. Dr. Philip quotes a similar case in the paper which I am now discussing. Knowing that a possible and not uncommon cause of acute general tuberculosis is the invasion of a pulmonary vessel by a caseous bronchial gland, I have a dread which I consider wholesome of any procedure which may cause mechanical disturbance of such a gland. The mishap, I admit, is extremely uncommon, but I maintain that its possibility ought to be taken into consideration before deciding to tap a case of pleural effusion in the early stage.

My impression is that early tapping does not always save time: a repetition of the operation is often necessary. Dr. Philip has enumerated other risks, and I agree with him in the opinion that they are almost negligible, but I think that they should not be absolutely ignored.

My own rules of procedure may be summed up as follows:

(1) If when the patient is first seen the effusion is so large that it is producing effects mechanically or becomes so during the period of observation, relief of symptoms should be given promptly by tapping, but aspiration should not be continued after the symptoms have been relieved.

(2) If the effusion is recent, serous, and not producing mechanical effects, the patient should be kept in bed for ten to fourteen days and the progress of the effusion watched.

(3) If during this period the effusion begins to produce mechanical effects, or if at the end of it there are no signs of its diminution in volume, some of it should be removed. The removal of a small quantity may be followed by the rapid absorption of the rest.

(4) Always hesitate to tap if the effusion is tuberculous, and especially if it is associated with pyrexia, unless you are certain that the cause of the pyrexia is not active inflammation of bronchial glands.

(5) In tapping never aspirate vigorously, and stop at once if the fluid becomes markedly blood-stained, if pain becomes acute, or if the patient begins to cough much.

Treated on these lines it is possible that the duration of confinement of the patient may be slightly increased, though I am not certain that this has been demonstrated, but the patient will not be submitted to the risks enumerated, which, though slight, are definitely to be feared.

The large majority of patients, moreover, will recover without the discomfort of the operation.

The removal of the fluid may be affected by means of a Southey's trocar and cannula, which acts by syphonage, and does not mechanically disturb the lung so much as the operation of aspiration. In practice there is always some risk of the ingress of air through the tube with the production of a pneumothorax. The air, however, is not under tension and is soon absorbed. I have seen the accident occur on several occasions, but never with any ill effects.

Sir James Barr recommended the removal of the fluid and its replacement by sterile air. I do not see any advantage in this procedure.

Aspiration by means of Dieulafoy's or Potain's apparatus is quicker than tapping by means of Southey's tubes, and if force be not used and no attempt be made to remove the whole of the effusion I do not think that this method is likely to be harmful, provided that the rules I have outlined be observed.

The site of election for tapping a pleural effusion is in the posterior axillary line at the level of the nipple, but the site of the puncture must vary with the size and position of the effusion. Before inserting the needle at any point it is wise to make certain that there is dulness and that vocal fremitus cannot be felt. A knowledge of the surface anatomy of the pleural sinuses is of great importance in determining the spot for puncture, as I pointed out in a former lecture.

I show you the instruments most frequently employed, but confidence in their use can be obtained only by practical experience.

November 4th, 1912.

ON SYPHILIS OF THE NERVOUS SYSTEM IN INFANCY, CHILDHOOD, AND EARLY ADULT LIFE.

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(*Concluded from page* 64.)

(8) Spinal Lesions.

Whereas spinal lesions are quite common in adults the subjects of the acquired disease, spinal lesions, apart from tabes dorsalis, appear to be comparatively infrequent in inherited lues. Dr. James Taylor, in his well-known book, 'Nervous Diseases in Childhood and Early Life,' makes scarcely any mention of lesions of the cord except those associated with tabes and some forms of pachymeningitis. Dr. Wilfred Harris, in his detailed article in Allbutt's 'System,' states: "Congenital syphilis may affect the spinal cord and its membranes as in the adult, and may produce symptoms soon after birth or later in life." Dr. J. Mitchell Clarke writes: "In hereditary syphilis the cord is occasionally affected, apart from the occurrence of tabes, and such affection may show itself in childhood or for the first time in an adolescent who has previously been in good health. The changes in the cord in new-born infants the subjects of congenital syphilis have been recently described by Toyofuku, and closely correspond to those found by Ranke in the brain in congenital syphilis." This writer quotes two cases that came under his notice. The first was a young man, æt. 21 years, who first manifested symptoms at the age of nineteen. He presented undoubted evidence of the inherited disease, and was admitted for failure of sight and weakness of the legs. Headache was intense. In addition to weakness of the right external rectus, nystagmus, and paresis of the left face, the patient had double optic neuritis. At first there was no paralysis of the legs, but later, after a period of distinct improvement with antispecific treatment, he returned with the signs of a transverse lesion of the cord. The subsequent progress of the case justified an exploratory laminectomy, "but the condition of the cord was evidently too extensive to be dealt with by operation." The patient died one month later, and at the post-mortem "the pia arachnoid was thickened and opaque at the base and over the cerebellum. The cerebral ventricles and iter were much distended and full of clear fluid. The spinal dura was externally normal. On slitting it up the pia arachnoid was thickened, gummy or jelly-like—adherent to the cord and also to the dura—over the cervical and lumbar enlargements, and over the most part of the dorsal cord. This change was most marked over the cervical enlargement, and here and over the lumbar one there was much injection of the vessels and the cord was softened."

The second case was that of a boy who showed marked signs of infantilism. He was much emaciated and could only say a few words on admission. He was almost imbecile. He could not stand or walk. The muscles were wasted from disuse and the legs were drawn up on the abdomen. There were no sensory disturbances and the reflexes were exaggerated. The liver was much enlarged. A small piece of the liver removed at operation showed the typical appearances of congenital syphilitic disease. Under a long course of mercurials and potassium iodide the paraplegia improved until "he was able to run about like a normal boy."

Singer also reports an instructive case of *acute paraplegia* occurring in a boy, æt. 12 years, the subject of inherited lues. The paraplegia developed rapidly and the child died seven weeks later. At the post-mortem gummatous meningitis and specific endarteritis were found. The sixth dorsal segment showed a definite focus of disease. Overgrowth of perivascular connective tissue rather than softening was conspicuous.

Whereas, too, acquired syphilis in the adult is responsible for a high percentage of cases of myelitis (80 per cent., Taylor and Buzzard) it does not appear to be a frequent lesion in the inherited disease.

Amyotrophic lesions analogous to those seen in the Aran-Duchenne type of progressive muscular atrophy are met with in tabes (Wilson, Léri and Marie). Further, in the adult, certain forms of *chronic and subacute poliomyelitis* are the result of acquired syphilis (Nonne and Hoffmann). I do not know of any similar lesion in a child the subject of the congenital malady. It appears that *acute poliomyelitis* and syphilis are in some cases related. Hoffmann (quoted by Harriss) has recorded an interesting clinical case of acute poliomyelitis occurring in a congenital syphilitic æt. 20 years. The paralysis appeared suddenly after some premonitory pains in the arm and back. Atrophic paralysis of the right arm and leg resulted. The deep reflexes were lost. There was no sensory or sphincter defect.

I have no instance to bring before you of gumma of the spinal cord, of the condition known as Erb's lateral and combined sclerosis, occurring in a patient the subject of inherited lues.

(9) Acute spinal lesions of the classical type whether they occur in infancy, childhood or early adult life, viz. acute poliomyelitis (with the exceptions noted above), acute generalised poliomyelitis, acute ascending myelitis and the con-

dition usually described under the title "Landry's paralysis" bear no relation to syphilis. The same remark applies to acute toxic polyneuritis. There is no reason to suppose syphilis plays any part in the production of the neuritic type of muscular atrophy, the peroneal type of Charcot-Marie Tooth.

In the only instance of *progressive spinal atrophy of infants* of the Werdnig-Hoffmann type which has come under my own notice, the blood gave a negative Wassermann reaction.

The myopathies.—Syphilis is not an ætiological factor in any of the more common or rarer types of myopathy which occur in infancy or childhood. The same remark applies to amyatonia congenita (Batten). In certain instances of myopathy syphilis may be an accidental association. Mckisack has recently reported the case of a young man, æt. 28 years, a plater, who developed a muscular dystrophy of the juvenile type (Erb). The patient's blood gave a positive Wassermann reaction, although he denied having had venereal disease. To my mind it would appear that syphilis predisposed to the development of a dystrophy with this distribution in an individual who had been "accustomed to severe muscular exertion, especially of the arms and back."

The prognosis in syphilis of the nervous system in the child.—In early life lesions of the nervous system, whatever their nature may be, are serious. Those occasioned by syphilis are still more so. The immediate as well as the future outlook is grave. In early life meningitis and hydrocephalus of syphilitic origin are especially fatal. If blindness already exists nothing of course can be done to make the optic nerves regenerate. The outlook in the diplegic variety is equally unsatisfactory. If the child lives, chronic imbecility is the ultimate state in the majority. Sudden death is liable to supervene at any moment. As in all delicate children, acute infections, pneumonia, measles, and scarlet fever prove fatal very readily. Much can be done for the cases of specific hyalitis if the condition is recognised early. Suitable treatment gives satisfactory results, but until the vitreous haze has cleared and an opportunity arises to examine the state of the optic discs and the surrounding retina too favourable a prognosis should not be given.

The type of meningitis which occurred in the child, Amelia W—, suffering with a mild grade of papillitis and cranial nerve weakness responds well to mercury and potassium iodide. Treatment, however, must be continuous, or relapse is certain to occur. Diffuse cerebro-spinal lesions, pseudo-tabes and the like, show remarkable improvement under efficient treatment. Where, however, considerable damage has already been done to the nervous structures I doubt if anti-specific treatment can do more than cause arrest.

Treatment.—Whereas all, or nearly all, observers are agreed that treatment with mercury and iodides is not applicable to definite cases of meta-syphilis, I am inclined to the view that in the early stages and in those cases where we are unable to decide for a time whether we are dealing with metasyphilis or conditions resembling it, temporary treatment with mercury and iodides is the correct plan to adopt, because whereas the cases simulating the para-syphilitic conditions are much benefited, the corresponding harm we may do is relatively unimportant. So far as I can see no harm has resulted in the cases of diplegia I have treated with anti-specifics. Where syphilis and epilepsy coincide in the same individual I am quite convinced that mercury, iodides and bromides are more beneficial than simple bromide medication alone. In my own hands in children who attend the out-patient department, grey powder and iodide of potassium have given satisfactory results. Inunction in the case of an out-patient is to my mind not so satisfactory and efficient a form of treatment as is the oral method. The parent will not, or cannot, give the adequate time and trouble. I prefer to keep intra-muscular injections for my adult patients. I have no personal experience of "606" and the more recent arsenical preparations in the treatment of children who are suffering from lesions of the central nervous system.

Summary.—(1) Syphilis produces lesions of the nervous system in young subjects with far greater frequency than is generally supposed.

(2) Whilst it is more usual to meet with lesions of the nervous system due to inherited syphilis, we must not lose sight of the fact that in a small percentage acquired syphilis is responsible.

(3) It is important to remember that the nervous lesions in acquired syphilis may occur very soon after the primary infection. In inherited lues, whilst most of the nervous lesions first manifest themselves within the first few months or even years, they may occur at any age up to puberty, at which time the para-syphilitic diseases are usually met with for the first time.

(4) If we admit that a Wassermann reaction of the blood, when positive (with well-defined exceptions), is conclusive evidence of syphilis, past or present, then we have in this reaction a very valuable method of examination, because a positive Wassermann reaction of the blood may be the only positive evidence we have of syphilis.

(5) A careful chemical and cytological examination of the cerebro-spinal fluid, especially with the application of a Wassermann reaction to this medium, is essential. It will, in a large percentage of cases, enable us to differentiate syphilis of the nervous system which is amenable to treatment from para-syphilis. Where syphilis appears to produce a primary neuronic decay or dystrophic condition in the nerve structures apart from any pronounced

meningeal changes, we must not expect to find changes in the cerebro-spinal fluid that we usually associate with syphilis and para-syphilis of the nervous system.

(6) Whilst the evidence is rather conflicting, it would appear that syphilis is responsible for a higher percentage of simple mental defect than is usually stated.

(7) Whilst syphilis is responsible for a high percentage of cases of interstitial keratitis it is the only cause of hyalitis in infants, a rare but well-recognised type of amaurosis in infancy.

(8) During the first few months of life syphilis is a not infrequent cause of meningitis and hydrocephalus. A lumbar puncture and a Wassermann reaction afford great assistance in the differential diagnosis.

(9) The part which syphilis plays in the production of diplegia is uncertain. The writer thinks there is evidence to show it is responsible in a few cases, more especially of the ante-natal type. It is difficult to prove or disprove an accidental association.

(10) Syphilitic epilepsy is infrequent. The writer is inclined to the view that, in infants especially, convulsions are more likely to occur in those who are the subject of inherited lues than in children who are otherwise healthy. In older children syphilis may be the sole cause of mental deficiency and fits of the idiopathic type. Fits of the Jacksonian type and the epileptiform seizures of general paresis are not included in the above statement.

(11) Whilst myelitis is a common lesion in adults who are suffering from acquired syphilis, spinal lesions apart from tabes appear to be infrequent in the subjects of inherited lues.

(12) The para-syphilitic affections of inherited lues closely resemble those seen in the subjects of the acquired malady.

(13) The morbid anatomy of inherited and acquired syphilis of the nervous system appear to be identical.

(14) The prognosis in diseases of the nervous system due to syphilis is grave. Early diagnosis is essential to procure a good result from treatment. In the Wassermann reaction and a careful examination of the cerebro-spinal fluid we have the most efficient aids to an early diagnosis.

Addendum.

Since writing the above, another case of hereditary syphilis with major epilepsy has come to my notice.

Case 13. *Violet C—, æt.* 10 *years. History.*—A fit three days ago. "Twitches all over; prostrate after the fit and dazed for two and a half hours; went to sleep." Sphincters not relaxed. Never any previous fits. Has had scarlet fever and "fluid on the lung." No recent injury or illness.

Family history.—Is the eighth child of a family of nine—five girls and four boys. Eldest 27 years, youngest 8 years. The mother, as a girl, had fits. One brother, æt. 23, had a fit when thirteen years old.

Present state.—The bridge of the nose depressed; dentition normal; well-nourished: looks healthy. There are no physical signs of organic disease in the nervous system. Examination of the fundus reveals undue pigmentation, but no choroiditis.

The *Wassermann reaction* of the blood is strongly positive.

Remarks.—Under treatment no further attacks, but severe periodic headaches. In all probability a case of idiopathic epilepsy occurring in a child the subject of inherited lues.

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A DEMONSTRATION OF GENERAL SURGICAL CASES.

In the Wards of St. Bartholomew's Hospital.

By C. GORDON WATSON, F.R.C.S.,
Surgeon to the Metropolitan Hospital.

ACUTE LUMBAR ABSCESS.

WHEN this child (æt. 6 years) came in a week ago it was thought that she had disease of the hip. The mother said the child was drawing up the left leg, but moving the other leg freely. A few days after admission a swelling developed on the back. You see that there is a diffuse swelling in the left lumbar region, extending from about the ninth rib to down below the crest of the ileum. It is a fluid swelling, and we have ascertained that it contains pus. Why is the thigh drawn up in this way, and what is the cause of the lumbar abscess? Is this abscess in connection with disease of the hip-joint, spinal caries, caries of one of the lower ribs, or with disease of the sacro-iliac joint, or the crest of the ileum? There is no pain on movement or swelling of the hip-joint. We do not think any bone is diseased. The X rays show no evidence of spinal caries or caries of the ribs. Is this an empyema which has pointed and extended into the subcutaneous tissue? Examination shows the chest to be normal. On referring to the temperature chart it becomes clear that the abscess cannot be classed as a "cold abscess"; the child is running a septic temperature varying between 99° and 101° F. There is a history of a fall, and these symptoms have come on consecutive to a fall. I have had the abscess punctured and some of the pus has been examined, and it gives a pure culture of *Staphylococcus pyogenes aureus*. I believe it to be *an acute staphylococcal abscess following on injury.*

These cases are not very uncommon, and it is a class of case which closely resembles acute osteo myelitis. A child has a kick on the shin and develops a high temperature, tenderness over the tibia,

and pus under the periosteum ; you open theab scess and obtain a pure culture of the *Staphylococcus pyogenes aureus*. In the same way a muscle may be ruptured, *e. g.* the psoas, and be followed by a similar train of symptoms and an abscess giving a pure culture of *Staphylococcus pyogenes aureus*. So, too, after subcutaneous injuries with hæmorrhage, there may be rise of temperature, pain, swelling and abscess formation due to the *Staphylococcus pyogenes aureus*. I believe this to be closely analogous to acute osteomyelitis. Mr. Moreton will open the abscess, sew it up again and apply pressure. If that does not cure we shall have to use a drain. The child is ill, but not severely so. The leucocytes number 18,000. You will note that there was no sign of the swelling until after the patient was admitted. The position of the hip suggests hip disease or a psoas abscess and is very misleading at first sight. No doubt it is a position of ease, but an unusual one for lumbar abscess. There is no actual contraction of the muscles.

Femoral Psoas Abscess.

Here is another interesting case, of a little girl who came in with a large abscess in the adductor region of the right thigh. You can see where the abscess was. She has now a small reaccumulation of fluid in the same situation. This hip was so contracted that when the patient's back was on the bed the thigh was flexed to less than a right angle and it could not be moved. Directly movement downwards was attempted up went the lumbar spine. On examination of this abscess it was found to be localised. On palpation over the psoas no swelling could be felt, but on turning the child over it was obvious she had spinal caries. As we are unable to move the child in this splint you must take it from me that she has angular curvature in the upper lumbar and lower dorsal region. This collection of pus in the thigh is due to a psoas abscess, and it has tracked down through the substance of the psoas under Poupart's ligament, on the outer side of the vessels, and has accumulated close to the attachment of the tendon of the psoas muscle. As a result of this abscess she developed such extreme contraction of the psoas muscle that her thigh was flexed to less than a right angle. A psoas abscess usually points close to the anterior superior spine, although it may run down under Poupart's ligament and point in the thigh, as in this case. In a few rare cases it passes backwards through the great sacro-sciatic notch. Not infrequently you may find an abscess in the region of the anterior superior spine, with a collection of pus below Poupart's ligament, and then you can often get fluctuation from one swelling to the other. It is unusual to find a large collection of pus which has come down the psoas, projecting into the thigh, and not to find also a collection of pus in the psoas muscle above Poupart's ligament. The abscess was incised. We adopted the precaution of not incising over the softened or inflamed area. The incision was made as low down as possible away from the inflamed area, the pus evacuated, and the cavity washed out with weak iodine. The incision was sutured and pressure applied. There has been some reaccumulation of fluid, so that the process will have to be repeated. You will note a difference between this and the last abscess I showed you with regard to the temperature, which in this case is normal : there is no septic infection and the abscess is a "cold abscess." With regard to the treatment of the contracted thigh, we have applied weight extension, maintaining the child steady on the bed by keeping a long Liston splint on the other side of the body. That is an essential point, especially in the case of children. We have gradually pulled on the thigh. Originally it was vertical, but now you see we have been able to get it to an angle of 45°. We have got rid of all the spasm of her psoas muscle and of other contracted tendons, and her thigh will now come down to the angle which you see, while her back is flat on the bed. Mr. Moreton has gradually lowered this splint day by day. It has now remained in this position for some days, and it has not been possible to lower it any more. We know what is preventing this leg from coming down. It is not muscular spasm, but fibrous contraction of the soft parts in the neighbourhood of the hip-joint. So the next thing necessary will be to tenotomise any contracted bands, and to manipulate the limb under an anæsthetic and then to re-apply weight extension. If fluid reaccumulates in the thigh, the abscess will again be evacuated and again sewn up. The reason we do not drain these cases is that secondary infection so frequently results from drainage, and then it becomes a difficult task to cure them. I do not scrape such an abscess but open and wash out thoroughly. If any

scraping is done it should be very gentle. If you scrape the wall of a psoas abscess at all extensively two things may happen: In the first place there may be troublesome hæmorrhage afterwards, and the blood may accumulate in the cavity of the psoas and cause trouble; and secondly, if you scrape the wall of such an abscess and remove the protective membrane, you may open up blood-channels to a general systemic tuberculous infection. I have seen a chronic tuberculous abscess scraped and tuberculous meningitis occur within three days, so that it is not advisable to scrape a tuberculous abscess in connection with the psoas muscle. I do not wash out with strong antiseptic solutions, but with saline or very weak iodine. Some surgeons inject iodoform emulsion in such a case after irrigation. I have found that the iodoform emulsion does not always get absorbed, and you may have to reopen the cavity to remove pledgets of iodoform which act as sequestra.

Multiple Teno-synovitis.

This patient is æt. 25 years. She has a swelling on the front of each wrist, and one on the dorsum of the left foot; there is nothing abnormal about the right foot. The malady is teno-synovitis, and it has existed for about three years. Obviously it is not traumatic, for it is multiple, affecting both hands and one foot. Her general health is quite good, and she has no disease of her joints. Such a condition as this is usually due to a very chronic form of tubercle. But it is very rare indeed to find the tendons of the foot affected. If you palpate these tumours you will feel that though fluid, there is a sensation of solid lumps in the cavity. If you look closely at this X-ray picture of the carpus you will see what appear to be a number of small holes in the carpal bones. But on more careful inspection I think you will agree that they are not holes, but are bodies superimposed on the bone. One on the lower end of the ulna is projecting beyond the free edge of the bone, and clearly cannot be a cavity in the ulna. On looking at the ulna of the opposite side you see the same condition of affairs, and over the unciform bone the same kind of body is projecting on to the base of the fifth metacarpal bone. So I think it is certain that those round bodies which you see are either melon-seed bodies or villous fringes. I do not know why they show so clearly in the X-ray picture. But we do know that these bursæ in connection with the tendon sheaths frequently contain melon-seed bodies. Here is a specimen which I removed some years ago from the dorsum of a girl's wrist. It was a recurrent bursa, for it had been opened on a previous occasion, and the tendon had been divided in taking it away. The tendon sheath during recurrence had become expanded into this cyst, which was full of these melon-seed bodies. These melon-seed bodies are tuberculous. One of those bodies was placed under the skin of a guinea-pig and resulted in general tuberculosis of the animal. The strong probability is that we are dealing with an exactly similar condition in this patient. So we propose that this patient shall have these bursæ in connection with the flexor tendons of the wrists and ankle dissected out. Although multiple teno-synovitis when not traumatic is usually tuberculous, it may be met in the course of gonorrhœa and syphilis, and is also described under the heading rheumatic, but we do not know what this term means pathologically. This patient has no disease of the joints in the immediate neighbourhood. The outlines of the bones of the wrist-joints are perfectly clear and distinct, and there can be no joint disease when you see such clearly defined outlines. If the synovial membranes between the bones were involved in disease there would not be such a clear definition, but a blurred outline.

Non-union of Fractured Femur.

This boy, æt. 12 years, fell down a month ago and broke his femur. He has been here three weeks. He did not come in for the first week. As he lies there with his everted thigh and shortened leg you will note that he has still a broken femur; it remains ununited in spite of weight extension and splints for three weeks. The X-ray picture shows you that it is a transverse fracture of the femur, with considerable overlapping. Owing to the fact that the boy did not come in until a week after the accident Mr. Moreton has not yet been able to get rid of all the overlapping due to the contracted muscles. There is some callus there, although the fragments are not in apposition. The question arises, How long are we to leave this boy in a splint with weight extension? Should he be subjected to an open operation? This time last year I had some cases in the ward of a similar

nature. Here is the skiagram of a boy who was about the same age as this boy, who came in with mal-union of the femur. You see that the bones were united at an angle of about 120°. I had to cut down and separate them and secure apposition by means of a plate. The result is good, and he made an excellent recovery. This radiogram, taken some months later, shows a straight bone with very little shortening. Provided we can secure asepsis, there is no doubt that we can get a good result in the case under consideration, but that is not so easy as you might think from published reports. This boy of course may have some muscle interposed between the fractured ends which will prevent bony apposition even when extension is applied and the bones manipulated under an anæsthetic. Personally I am in favour of continuing treatment by weight extension for some weeks longer, because the functional results in cases of fractured femur in children are astonishingly good. Growth rapidly corrects any deformity. I had an opportunity a little while ago of seeing a good many results of fractured femur at the Metropolitan Hospital. These were being examined by a Commission of the British Medical Association with regard to the treatment of fracture of the femur. Among the young, after an interval of a few years it was very difficult indeed to tell which leg had been fractured; in some you really could not detect the past existence of a fracture. So I am loth to cut down on that bone at the present time, because if I can get rid of most of that shortening and get more callus to form, even if the ends are not in accurate apposition, the functional result is likely to be very good. He should not be left with more than half an inch of shortening if it can possibly be avoided, and, of course, he cannot be left with his bone ununited. If I do cut down the incision will be made on the outside and the ends of the bone will be pushed out through the wound, and if there are any soft parts intervening that will be attended to. In the case of a transverse fracture, it is a simple matter to get the ends into apposition if they are first pushed out through the wound, then made to impinge and pushed back while in contact. Having got the bones back they will be held in position by these long forceps which Lane invented, and a plate screwed on. Here I show you the skiagram of another man who was in at the same time. This thigh remained ununited, because soft parts got in between the bones. We treated him with weight extension and splints for seven or eight weeks without the slightest attempt at union taking place, and then I cut down and found soft tissues between the fragments. I removed these and applied a plate. This radiograph shows the good result. The weight we use is generally half a pound per year of the patient's age up to twenty years of age. It varies somewhat with the individual: a strong healthy boy would probably require more, and a weakly boy less. This boy is æt. 12 years and the weight used is 6 lb.

Recurrent Tuberculous Disease of the Knee.

Here is a very ordinary case of tuberculous disease of the knee. The disease here is in the synovial membrane, not in the bone. You can tell this from the X-ray picture, because the epiphysial lines are normal, there is no cavity in the epiphysis, and the outline of the synovial cavity of the joint is blurred and blotchy, which is typical of tuberculous disease. I show him because I was exhibiting some of these cases to you yesterday, pointing out the deformity which occurs and the method of treatment, and I called your attention to the danger of recurrence if treatment was relaxed. This boy was in the hospital in November, 1911, and we treated him for his tuberculous knee by weight extension. His leg was put up on a Marsh's knee-splint. The leg was kept horizontal for a certain time after flexion had been relieved, until we were sure that the spasm was relieved. Then we put him into plaster-of-Paris and he attended the out-patient department. His plaster was taken off and he was ordered to come back to have another plaster put on, but he did not return. He has now come into hospital again a year later because the flexion has recurred and the joint is hot. We have now to treat him again first by weight extension. If the patient is able to afford it, the best splint to apply after weight extension has overcome the contraction is a leather splint, with iron supports to the heel to prevent the splint sliding down. The splint can be taken off for washing and cannot be broken.

Dislocation of the Semilunar Bone of the Hand.

This patient, a man of middle age, fell a distance of twenty feet on June 12th, and received the

force of the fall on his extended wrists. He was unconscious and was admitted to the Seamen's Hospital, where he remained sixteen days. He had a wound in the right wrist, which was sutured, and he attended there afterwards as an out-patient. Both wrists remained stiff and painful, the right being much worse than the left. There is still some stiffness in the left wrist. But apparently he has had no gross injury to that wrist, that is to say, no fracture or dislocation. Here is a skiagram of the left, and apparently, as far as the bones are concerned, it is normal. Here is the skiagram of the right side. At first sight it appears from the skiagram as if there was some injury to the radius. But if you look more closely and trace out the bones of the carpus you will see that the scaphoid is not normal, and that at the side of it there is a little knob of bone which is not normal. It looks as if the tubercle had been knocked off the scaphoid. You can see the trapezium, the trapezoid, the os magnum, and the unciform. And if you come back to the other row you see the scaphoid, cuneiform and pisiform, but no semilunar. So there has been a dislocation of the semilunar as a result of a fall on the wrist. The semilunar bone has been shot out and wedged up against the front of the radius. If you examine his wrist you are able to feel where the semilunar bone is. That has been there three months. Fracture of the scaphoid is a common accident as a result of a fall on the wrist, but dislocation of the semilunar is rare. It is a nasty sort of case to have to deal with in private, especially if you have not an X-ray picture, because there is no cure without operation. I shall remove it. If I tried replacement I might replace the bone but not the ligaments, and it would be sure to come out of place again. Moreover the removal of the bone will not impair the use of the hand. I have removed the scaphoid for fracture and there has been no apparent loss of function; the other bones take on the work. I think it is wise, if a fracture of the scaphoid or other carpal bone gives rise to symptoms, to remove it at once, because its removal does not result in any definite disability. Union seldom results and osteo-arthritis frequently follows if it is not removed.

Inoperable Malignant Disease treated by "606."

I now want to show you some photographs, because although I have not the patient here now, I referred to him at consultations yesterday. Here are the photographs taken of the man before and after treatment by "606." He had malignant disease of his thyroid gland. The growth was first removed in 1905, and in 1909 a recurrence was taken away. It recurred once more and was removed in 1911. Recently he returned with a recurrence on the side of his neck, which was inoperable, and he was suffering a good deal of pain. I thought I would give him an injection of salvarsan, in order to see if I could relieve the pain. I did that, and within a few days the lumps rapidly became smaller and the pain disappeared. He had only one injection, and this was responsible for very definite improvement. You can see the effect on the tumour from the photographs. I do not for a moment suggest that I have cured his malignant disease, but I have made his life more bearable and taken away his pain for the time being. If that swelling gets bigger again, as I expect it will, and he comes to me, I shall give him another injection of "606." I gave it because I heard of its successful use in Germany for inoperable sarcoma. I tried it first on a case in private, a case of inoperable sarcoma of the ileum. There was some relief of pain there but no alteration in the size of the swelling. I do not know of others having used it in this country for inoperable cancer. In inoperable cases it is of inestimable value to be able to do something, however little, for the relief of the patient's distressing symptoms.

Ileo-Sigmoidostomy for Carcinoma of the Colon.

This man came in on May 3rd, under Mr. Waring, with intestinal obstruction. Mr. Waring opened the abdomen and found obstruction in his large intestine. A lump existed in the region of the splenic flexure, and he performed transverse colostomy. The patient went to the Convalescent Home at Swanley, and returned here on July 31st for something more to be done. On August 26th I opened the abdomen in the middle line. The reason I opened the abdomen was to see in the first place whether, the colostomy having been established and the obstruction relieved, the growth was removable. And secondly, if the growth was not removable, to short-circuit the intestine so that eventually the colostomy could be closed. I found

that the growth was fixed and clearly was not removable. I then anastomosed the termination of his ileum into his sigmoid flexure, *i. e.* I performed ileo-sigmoidostomy, and the result of that has been very satisfactory. He now passes his motions by the rectum as well as by the colostomy. It is not without danger to make an incision in the abdomen in the neighbourhood of a colostomy wound. We did our best to avoid infection of that sort by careful covering up of this wound, first with dressing and then with layers of strapping over it, before opening the abdomen. He had a little skin infection in the upper part of his wound. Now the ileo-sigmoidostomy is established, the next thing to do is to close the colostomy, and that will have to be done soon. It is important to make a long anastomosis through the peritoneal and muscular coats, otherwise you are very liable to get a kink at the opening. My line of anastomosis between the ileum and the sigmoid was 4 in. or more, whereas the opening in the bowel would not be more than 1½ ins. So on either side I had quite three quarters of an inch of bowel joined together, to prevent any kink occurring by the ileum falling down into the pelvis. Another point to remember is that if you perform ileo-sigmoidostomy for obstruction of the large intestine you may not altogether relieve the obstruction, because the ileo-cæcal valve being perfect will still allow of obstruction behind the growth. Perforation of the cæcum may occur in spite of ileo-sigmoidostomy. In a case upon which I operated that did occur. The patient had carcinoma in the transverse colon due to obstruction. The growth was inoperable, and I wished to avoid cæcostomy. I thought then it would suffice to short-circuit ileum and sigmoid, in order to relieve the obstruction. Relief was only temporary and shortly afterwards perforation of the cæcum occured. The cæcum, being distended at the time of the operation, remained distended, because the tension of the ileo-cæcal valve prevented any back-flow.

Fracture of the Ilium.

This man fell on his hip off a scaffold a distance of twenty feet. Under these circumstances you would expect a fracture of the great trochanter, an impacted fracture of the neck of his femur, or a fracture of the pelvis, the head of the bone being driven through the acetabulum. Those are the common accidents in connection with heavy falls on the hip. In his case the great trochanter and femur are uninjured, but he has a rather unusual fracture, namely, a fracture of the crest of the ilium commencing one and a half inches behind the anterior superior spine and extending forwards. You see there is a good deal of bruising over that region. Not very long ago we had a patient in the hospital who had a similar but less fortunate accident. He drove the head of his femur through the acetabular cavity, and so fractured his pelvis. It was found at the autopsy that he had a very extensive hæmorrhage into the cellular tissue of his pelvis on the other side of his acetabular cavity, where the head of the bone had been driven through, and the head of the bone was close up under the peritoneum. Infection followed the hæmorrhage probably *viâ* the intestines and he died of acute pelvic peritonitis.

Malignant Disease of Glands in the Neck.

Here is a man who has had part of his tongue removed by Mr. Waring. In December, 1911, he had an extensive operation, as you can see from the scar, to clear out the glands in his neck. Now, nearly a year after that operation, he comes up with a large recurrent mass, a secondary deposit in his neck, presumably in the lymphatic glands underneath the sterno-mastoid, extending backwards into the posterior triangle. There is pressure on the small occipital nerve causing pain over the skull. There is no recurrence in his mouth, and the half tongue which was left him moves quite well. I do not think it is worth while in this case to do any further operation. That gland is very fixed, and it must be adherent not only to the carotid sheath, but to the bodies of the cervical vertebræ, and it must be close to the cervical sympathetic also, in addition to being close to the base of the skull. I do not think one could satisfactorily remove that without damaging some of the neighbouring structures. This is again the kind of case on which I think one might try the effect of giving "606." It is interesting to know that he has that recurrence in his neck in spite of the fact that he has had such an extensive dissection of his glands. We know Mr. Waring does these operations very thoroughly, and there is no doubt he would clear out everything which could be reached. I had a man with carcinoma of the tongue. I removed his tongue and the glands from both triangles of his neck; I not only cleared out the submaxillary triangle, but I made the incision which the late Sir Henry Butlin made, straight down the sterno-clavicular joint, with a transverse incision from the upper border of the thyroid cartilage to the symphysis menti, and cleared out not only the lymphatic glands which were palpable and visible, but all the deep cervical fascia, and, in fact, everything except nerves and vessels and muscles. I think that operation was as thorough as anything could be for glands in the neck. Yet in three months that man returned with a recurrence in his neck. Carcinoma of the tongue does certainly

vary in its degree of malignancy in an extraordinary way. The growth on the tongue was very small, and my operation in the neck was meant to be rather a protective operation than one for removal of disease. But it proved to be absolutely futile.

Large Epithelioma of Lip.

Here is a man, æt. 75 years, who has come here with epithelioma of the lower lip, which has been growing about twelve months. There is nothing special about his case. It is a typical squamous-celled carcinoma of the lower lip, and there are no palpable glands in his neck. With regard to the treatment of that case, he will have a V-shaped incision made in his lower lip. When that is done it will leave him no mucous membrane for his lower lip, and he will have microstoma or small mouth, consisting of a doubled-in upper lip. In order to improve upon that the cheek on either side can be split, and the mucous membrane of the cheek sutured to the skin of the cheek. In this way you provide him with a mouth which is double the size it otherwise would be, and you then remove a V-shaped piece involving the whole lower lip and draw the two sides together. The lateral gaps then open out in circular form and constitute a lower lip. I have done this before, and it gives a very satisfactory mouth. I do both procedures at the same time. I would like to say a word about the use of radium in these cases. Two years ago I had a man with epithelioma of the lip about the same size as this one, and a colleague of mine, who was interested in radium, suggested that I should treat it with radium rather than by operation. Under radium the growth became very much smaller, and the man was exhibited at the Royal Society of Medicine, with photographs, as a case indicating the value of treatment of epithelioma of the lip by radium. The improvement continued for some months. But after a time the epithelioma began to suppurate extensively, and the radium applications had to be stopped. From the time of the onset of the suppuration improvement ceased and the growth recommenced. But now, instead of the tumour growing, as is usual with these epitheliomata of the lip, very slowly, it grew with great rapidity, and the case was handed back to me and I excised the lip. Within three months from that time I excised a recurrence from the lip. Three months after that he returned with still another recurrence, and with the remains of the lip adherent to the lower jaw. I had then to remove not only what remained of the lip but a portion of the lower jaw also—a mutilating and extensive operation, which included a big dissection for glands in the neck. Therefore I do not think it is worth while to treat epithelioma of the lip by radium under the assumption that you will save the patient from surgical mutilation.

November 11th, 1912.

DEMONSTRATIONS ON METHODS OF INVESTIGATING GASTRIC DISEASES.

At St. Bartholomew's Hospital.

By W. LANGDON BROWN, M.D., F.R.C.P.,
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III. The Diagnosis and Treatment of some Organic Diseases of the Stomach.

Gentlemen,—I concluded the last lecture by considering the treatment of hyperchlorhydria, and I emphasised the fact that it was necessary first to know whether the case was one of simple hyperchlorhydria. The principal things which we have to exclude are, ulceration in the stomach or duodenum, gall-stones, which are often latent, chronic appendicitis and colitis. The examination of the stools for occult blood by the benzidine test, which I showed you last time, will be an important help in excluding ulceration. Secondly, examination for zones of hyperæsthesia is very useful. The patient lies on his back with eyes closed, and a pin is drawn lightly over the abdomen, from above the costal margin down towards the pubes, first on the left side and then on the right. He is directed to tell you as soon as he feels the pin to be sharper. Having determined the upper limit of hyperæsthesia, then start from below and work upwards in the same way. Do this in several vertical lines. In that way you may be able to map out a zone on the left side including the umbilicus, sloping slightly upwards towards the axilla. That corresponds to the distribution of the tenth thoracic segment, and is strong evidence of the stomach being the source of the trouble. The existence of epigastric tenderness and this definite zone of hyperæsthesia is suggestive of there being ulceration. On the other hand, if the trouble is referred from the gall-bladder or the appendix the zone of hyperæsthesia will be in the upper quadrant on the right in the former case and in the lower quadrant on that side in the latter. The absence of definite zones of hyperæsthesia is a point against an organic basis for the hyperchlorhydria. Large quantities of mucus in the stools will suggest the existence of mucous colitis. If that is present the ordinary methods of

treating hyperchlorhydria will probably not be nearly so successful, and attention must be directed more to the condition of the bowel.

Gastric ulcer and its sequelæ.—The orthodox method of treatment of gastric ulcer where there has been recent hæmatemesis is absolute rest, ensured by morphia hypodermically, and more recently half a drachm of liquor adrenalin chloride in half an ounce of water has been given by the mouth. This can be given at two-hourly intervals without risk, because its action is purely local; it is not absorbed from the alimentary canal, so that while constricting the bleeding point it does not tend to raise the general blood-pressure. After about twenty-four hours' rest the usual practice has been to feed by the rectum. But there is no satisfactory evidence that rectal feeding is efficient. I have carried out a series of experiments on patients to whom the following nutrient had been given four-hourly: milk four ounces, plasmon one to two drachms, dextrose one to two drachms. One drachm of liquor pancreaticus is added and it is kept at a temperature of 37° for twenty minutes, when it is administered slowly by gravitation enema with the addition of 5m tincture of opium to increase the tolerance of the bowel. The bowel is also washed out night and morning by a simple saline. The most reliable indication we have of the nitrogen in the diet being assimilated is that the nitrogen in the urine increases. Many of the observations on the efficiency of rectal feeding have been based upon recovery methods, that is to say the washings from the bowel are estimated, and it is assumed that the difference between the nitrogen in the washings and the nitrogen in the administered nutrient represents the amount absorbed by the body. But that is a fallacious test, because it is very difficult to wash out all the unabsorbed material. And the fact that nitrogen has been remaining in the bowel and been broken down into putrefactive bodies is not satisfactory evidence of its assimilation. In my experiments I compared the nitrogen output of patients on nutrient enemata with that of patients who were having nothing but salines *per rectum*. Fig. 3 is from a patient on salines, and the nitrogen excretion follows the curve which is seen in a healthy fasting individual. It falls rapidly, starting from nearly 13 grammes of nitrogen per day to about 5, and at that point it remains. Fig. 1 from a patient who was given salines for a few days, and then nutrients for a few days. Here it starts at about the same level, 11 grammes, and falls down to 6, and remains there until mouth-feeding is begun. Compare that again with Fig. 2 from a case where nutrients have been given from the start. The average output of nitrogen started on a little over 10 grammes, and fell to a point

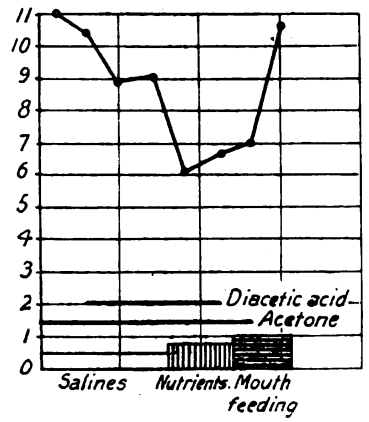

FIG. 1.—Nitrogenous excretion on rectal salines followed by nutrients.

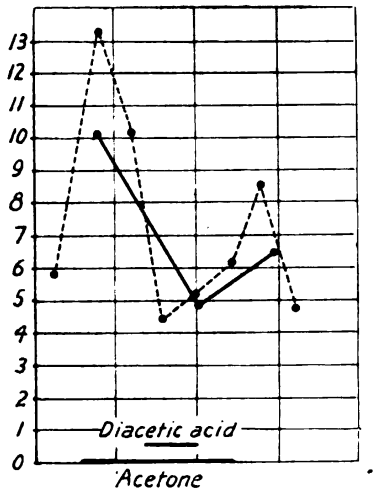

FIG. 2.—Nutrient enemata throughout. The dotted line represents the daily excretion of nitrogen, the continuous line the average of three-day periods.

between 5 and 6, and remained at about that level throughout the whole time nutrients were used. From this experiment there was no evidence that the nitrogen was used to any extent. If you wish to urge that the curve is slightly higher in the cases where the nutrients were used I would concede that there was a difference of possibly one gramme. But do you think it is worth while to subject the patient to all this inconvenience for the absorption of one gramme of nitrogen?

We might expect sugar to be absorbed better

than protein by the rectum. One of the best indications of the absorption of sugar is the abolition of acidosis. All rectally fed patients develop diacetic acid and acetone in their urine within forty-eight hours, just as a fasting person does, and you will see that the diacetic acid persisted in Fig. 1 even though nutrients were given. In one case (Fig. 2), which I have selected as favourable to nutrient enemata, diacetic acid was present only for a short time. In another case (Fig. 3) dextrose was given on only two days, but it was not able to abolish the acidosis. So the outcome of these experiments seems to be that sugar is absorbed to a certain extent by the rectum, but not always in sufficient amount to abolish acidosis. The

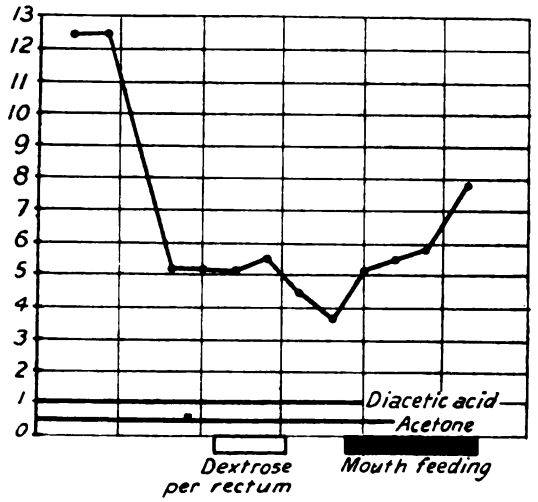

FIG. 3.—Rectal salines followed by salines with the addition of 5 per cent. dextrose.

assimilation of 30 grm. of sugar a day is sufficient to abolish acidosis (Halpern), therefore we must conclude that in these cases less than 30 grm. of dextrose was absorbed.

Thirdly, as to fats. I had an opportunity last year of examining a case under Dr. West's care of chyluria due to infection by filaria. The patient always passed a considerable quantity of fat in his urine on an ordinary diet. On a fat-free diet the amount of fat in the urine fell very considerably, but he was so sensitive to the addition of fat that giving him milk in his tea would lead to a distinct increase of fat in the urine. After he had been on a fat-free diet I gave him on one occasion 120 c.c. of pancreatised olive oil made up with 80 c.c. of 2 per cent. sodium carbonate. On another occasion I gave him one pint of pancreatised milk *per rectum*. In neither instance was the fat in the urine increased at all. It is hardly credible, then, that the fat administered in a nutrient enema can be of any service. In the case from which Fig. 1 is taken a somewhat crucial test was employed. After the patient had been on salines for some days he was given the standard nutrient for two days, and then he had the same amount of food given him by the mouth. You notice that the starvation curve persists until the mouth-feeding is resumed, when the nitrogen promptly begins to rise. Here we have a comparison between the effect of a diet given by the mouth on the one hand, and by the rectum on the other. I do not say that these experiments show that no absorption from the rectum occurs. A large enema can be carried through the ileo-cæcal valve, but how much of such an enema will be retained by the large bowel is, I think, doubtful. It is in the small intestine that absorption mainly occurs. What I do say is that these experiments show that nutrients as ordinarily given are of very little benefit indeed. Personally I do not think it is of any use to do more than give saline with 5 per cent. dextrose, which will yield all the advantages to be obtained by rectal feeding. Not only do I believe rectal feeding to be inefficient, but it has certain obvious drawbacks. It is very difficult to keep the patient in a cleanly condition; also it leads to a certain amount of secretion of gastric juice, which, being poured out into an empty stomach, will help the further digestion of the ulcer. Next, it produces considerable thirst, but this can be relieved by giving rectal salines as well. Fourthly it is liable to produce parotitis, due to septic infection from the dry mouth—a similar condition to that sometimes seen in typhoid fever. This risk can be reduced by giving the patient something to suck. During the latter part of the time that I was using rectal nutrients, I was accustomed to let patients have a bismuth lozenge to suck so as to keep the salivary glands active, and also to neutralise the acidity of any gastric juice which was formed. And certainly this made it easier to keep the mouth in a satisfactory condition. Another objection is that there is sometimes persistent vomiting. Unfortunately this is usually taken as evidence that the patient cannot stand food by the mouth, but it is apparently due to acidosis from starvation, and will persist until mouth-feeding is resumed. Lastly the

pronounced subnutrition induced by rectal feeding is very unfavourable to the healing of the ulcer and to the general condition of the patient, who is already anæmic and has been depleted by hæmatemesis. In 1901 Lenhartz advocated the practice of immediate feeding by the mouth in gastric ulcer. He pointed out the drawbacks of the ordinary plan of rectal feeding, and he maintained that the principles which should guide us in the treatment of gastric ulcer are (1) administration of sufficient food to maintain the patient's nutrition, but at the same time to prevent distension of the stomach, which would occur if a quantity of milk adequate to maintain nutrition were given; (2) a diet rich in protein, which fixes the hydrochloric acid by combining with it, and thus prevents its influence in continuing the ulcerative process. Bolton has shown clearly that hyperacidity is a potent factor in preventing the healing of gastric ulcer. Lenhartz thought these indications were best fulfilled by giving small quantities of beaten-up egg and milk, both of which are kept iced. In that way a diet rich in albumen is obtained and distension of the stomach avoided. His plan is to feed the patient by teaspoonfuls every hour from 7 a.m. to 10 p.m., allowing rest throughout the night. Table I gives the original Lenhartz dietary, and Table II shows what that dietary means in teaspoonfuls per hour for the first six days. On the first day the patient is to have two eggs and 200 c.c. of milk, and that means he has two teaspoonfuls of beaten-up egg and four teaspoonfuls of milk every hour. Sugar is added to the eggs on the third day. On the sixth day he gives some raw scraped beef. I think the creatin in the beef is an objection, as it tends to stimulate the secretion of acid, and therefore, in my cases I have substituted an equivalent amount of cooked minced chicken. By the seventh day he is taking a little rice, and on the eighth day some soaked rusk. On the tenth day he is having raw ham, according to Lenhartz. That probably suits the German patient, but the English patient does not relish it, and I have simply increased the amount of minced chicken. He recommends an ice-bag being applied to the epigastrium, but I have not often used that. He also recommends that the bowels should not be opened at all during the first week, in order to avoid peristalsis and to promote absorption of the outpoured blood by the intestines, and then if necessary to use a small glycerine enema or one of plain hot water. I have not allowed my patients to go as long as that without opening the bowels because of the amount of straining which is required when constipation has lasted so long. He claims as advantages for this method of treatment that the recovery is more rapid, and that it does not deplete the patient because the food supply is sufficient throughout. The sour regurgitation subsides, vomiting and bleeding cease more quickly and relapse is less frequent, while pain ceases promptly, and morphia is seldom required. Moreover, it is possible to

TABLE I.—*Original Lenhartz Dietary.*

Days	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Eggs	2	3	4	5	6	7	8	8	8	8	8	8	8	8
Sugar, with eggs (in grammes)	—	—	20	20	30	30	40	40	50	50	50	50	50	50
Milk (in cubic centimetres)	200	300	400	500	600	700	800	900	1000	1000	1000	1000	1000	1000
Raw scraped beef (in grammes)	—	—	—	—	—	35	70	70	70	70	70	70	70	70
Milk rice (in grammes)	—	—	—	—	—	—	100	100	200	200	300	300	300	300
Rusk (soaked), 1 piece = 20 grammes	—	—	—	—	—	—	—	1	2	2	3	3	4	5
Raw ham (in grammes)	—	—	—	—	—	—	—	—	—	50	50	50	50	50
Butter (in grammes)	—	—	—	—	—	—	—	—	—	20	40	40	40	40
Represents in calories	280	420	637	777	956	1135	1588	1721	2138	2478	2941	2941	3007	3073

TABLE II.—*Diet during first six days of Lenhartz treatment.*

Day.	Eggs (drachms per hour).	Milk (drachms per hour).	Sugar (*per diem*, added to eggs).
1	2	4	—
2	3	6	—
3	4	8	1 oz.
4	5	10	1 oz.
5	6	12	1½ oz.
6	7	14	2 oz.

treat anæmia earlier, and there may even be an increase in body weight during the first week.

One of the questions which will naturally be asked about this treatment is, what influence has it on the recurrence of hæmorrhage? Lenhartz found that in the hundred cases treated before he introduced this method recurrence took place in 20 per cent., but after introducing this treatment recurrence occurred in only 8 per cent. The method has been taken up on the Continent to a considerable extent and also in America, but up to a few years ago it had made very little headway in England. There seems to have been considerable prejudice against it. At last, however, people are beginning to realise that it is an important advance, and it is now rapidly becoming the orthodox method. I will give the results in my first thirty hospital cases, none of which have been treated during the last twelve months, so as to give some chance of tracing whether there was recurrence. But we know how difficult it is to do this in the case of hospital patients. I have only included the hospital cases, for I only saw once or twice with the medical attendant the private cases, and I have not accurate notes of them. There had been recent hæmorrhage in seventeen out of the thirty cases. In one case it is probable that the hæmorrhage was from cirrhosis of the liver, but I could not be certain as to the source and I have included it. The treatment was quite satisfactory in twenty-two cases. There was recurrence of the hæmorrhage in four. I will analyse those four cases a little further. In one there was recurrence subsequently with nutrients just as occurs with Lenhartz treatment. In another the recurrence was very slight, amounting to the regurgitation of a drachm of blood. In a third case with a history of sixteen years recurrence had previously taken place on nutrients. Gastro-jejunostomy was performed, and the hæmorrhage recurred a few months after the performance of that operation. Finally an ulcer surrounded by dense adhesions to the liver had to be excised, and the patient made a good recovery. In another case recurrence took place and gastro-jejunostomy had to be performed for a chronic ulcer. Unfortunately the patient died of peritonitis, the direct result of operation. As to the four which I have classified as unsatisfactory, one discharged himself on the fourth day—a thing which is common enough in patients treated by nutrients. In one case there was mucous colitis also; as I have already said, with this complication the treatment of the gastric condition, which I believe to be secondary, is often of no avail. Another case was that of a neurotic alcoholic lady on whom I tried it simply because no other treatment of her gastric pain had done any good. Ultimately she demanded an operation, but nothing could be found wrong then. The pain has recurred since the operation, and it will return as long as she continues her alcoholic habits. In the fourth case, although there was great temporary relief the X rays showed an hour-glass stomach, for which operation had to be performed. Here, too, there were dense adhesions, and a gastric fistula formed with fatal results. Clearly the fatalities had nothing to do with the Lenhartz treatment; they were both in cases of long standing, and certainly would not have been avoided by rectal feeding.

On the whole I have been most favourably impressed by the method, and I am supported by my house-physicians and the sisters in charge of the cases, who would much rather have to deal with a case treated on this plan than on the other, avoiding as it does the misery of starvation and all the discomforts of rectal feeding. The method is peculiarly suitable for the conditions of private practice. Nutrient enemata require time and skill in their preparation. We cannot expect them to be given properly except by a trained nurse, whose services cannot always be afforded. But in this method we have a simple, safe, and effective treatment, which is free from any disagreeable features.

Perigastric adhesions.—A complication or sequel of gastric ulcer which may give some trouble is perigastric adhesion. The following points in a case should suggest that we have perigastric adhesions to deal with. There will generally be a history pointing to gastric ulcer or to gall-stones. Local tenderness is very frequent. The pain is generally chiefly influenced by the position of the patient, and very little by diet. Vomiting is not a common symptom. The X-rays will prove, as I showed you in the first lecture, that the stomach is in an unusual position, and with change of posture or with contraction of the abdominal walls it does not move as freely as it ought. The influence of posture on the pain is very important. That was marked in one case of mine, where the patient was clear that the pain she now had was different from the one she experienced when she had gastric ulcer. When she was at rest

in bed there was no pain at all, but as soon as she adopted the upright posture the pain recurred. A skiagram showed the stomach pulled up and a little to the left. A test-meal showed an almost normal result. All this pointed to perigastric adhesions rather than active ulceration. Mr. Gordon Watson operated and found there were adhesions between the stomach and the abdominal wall, in the position in which the pain was felt. What we were not prepared to find was that the anterior abdominal wall formed the floor of an ulcer, that is to say, the stomach had perforated, but being adherent to the abdominal wall no further mischief had occurred. She made a good recovery. Such adhesions occur more commonly between the stomach and the liver or pancreas.

Dilated stomach.—Next as to dilated stomach. I would remind you of the important differences between the atonic and the obstructive form of dilatation. Atonic dilatation is found to be a commoner condition than was generally supposed before X-ray methods of stomach examination were introduced. There is sometimes a long history of chronic gastritis. In several of my cases there had been typhoid fever before the onset of the symptoms. I have met with instances, and I have heard of others in which both father and son suffered from it. One of the most characteristic symptoms, which can generally be elicited if you inquire for it, is that the patient feels full up as soon as he starts eating. The explanation, I think, is that given by Hertz. He points out that both the atonic stomach and the contracted stomach give rise to this sensation. The contracted stomach feels full up because it cannot expand. The atonic stomach feels full up because it is already dilated, and the normal mechanism, relaxation of the stomach as it fills, to avoid a rise of intra-gastric pressure, cannot take place. As soon as food is taken into this relaxed stomach the pressure begins to rise and the patient feels full. After taking food there is a general sense of misery and discomfort rather than actual pain. The patient frequently complains of a disagreeable taste in the mouth, and there is often a brown fur on the back of the tongue. This is probably due to regurgitation of the contents of the stomach through a relaxed sphincter. There is seldom vomiting. On examination you will very commonly find pyorrhœa. On inspection of the abdomen there may be some dropping of the stomach, a hollowing out under the costal margin and a fulness below and to the left of the umbilicus.

A succussion splash which is elicited only on deep palpation is only significant at a time when the stomach should be empty. A superficial splash has some significance. It is suggestive of atony, but it is sometimes distinguished with difficulty from the splash obtained in the colon. And we must remember that neurasthenics and hypochondriacs have an extraordinary power of producing splashing by vigorous contraction of the abdominal walls. The two halves of a seidlitz powder dissolved separately in water and swallowed produce a distended resonant area distinctly lower than it should be. The test-meal often shows diminished hydrochloric acid, but by no means always. Examination with X rays after giving bismuth is of great value. As I said in the first lecture, in a dilated stomach which is atonic we have a shadow reaching some distance below the umbilicus with a broad meniscus at the bottom; peristaltic waves are feeble or absent. This is not the type of case in which gastro-jejunostomy will be of real service unless there is so much dropping that there is actual kinking of the pylorus. Such kinking really adds an obstructive feature to the atonic case, and then there may be some purpose in gastro-jejunostomy. For let me impress upon you that gastro-jejunostomy is an operation which only gives its best results when there is evidence of obstruction. After a gastro-jejunostomy the food still continues to pass through the pylorus, and quite a considerable degree of obstruction must exist before the ostium or new orifice is used as the point of exit. Otherwise if the patient swallows a dilute solution of bismuth while standing in the upright position it does not pass through the ostium, even though that ostium may be at the most dependent spot. Gastro-jejunostomy is not a drainage operation. The contents of the abdomen are under conditions of hydrostatic pressure transmitted equally in all directions, and the onward progress of the contents results from local alterations in the pressure produced by muscular contractions. In the ordinary way the muscular contractions are sweeping across towards the pylorus, and therefore as long as the pylorus can, it will be the site of exit of the gastric contents. There is very little use, then, in attempting such an operation in the case of atonic dilatation. In

obstructive dilatation, on the other hand, the operation will give excellent results. The characteristic symptoms are vomiting of large amounts and steadily increasing discomfort between the attacks. After the stomach has been emptied by a large vomit the patient is comparatively comfortable for a few hours, and then discomfort begins and increases until it culminates in vomiting again. A point of practical importance which I have found is that the vomit is highly acid, if the obstruction is not of a malignant nature. Ordinarily, though the secretion of gastric juice may be normal or even slightly hyperacid, the vomit will not show free hydrochloric acid because the conditions which excite vomiting are almost certain to have previously inhibited gastric secretion. But when there is obstruction to the pylorus the vomiting occurs from quite a different reason. Secretion has been taking place as usual, but onward progress and consequent neutralisation is delayed so that the vomited contents will be highly acid. This point helped very much in the diagnosis in the case of a young lady who had had symptoms of gastric ulcer on two occasions some years before. She now had attacks of vomiting. Between those attacks she seemed quite well. The vomit had an acidity equal to ·292 per cent. HCl, which is distinctly higher than that shown by the ordinary test-meal removed with the tube. So that I felt sure that, whatever else there was, she had obstruction to the pylorus, which X-ray examination, and finally operation, showed to be the case. When there is obstructive dilatation, X rays show vigorous peristaltic waves. You see the waves twisting about in the stomach, but you do not see the onward progress of the bismuth into the duodenum. We must distinguish between dilatation and the condition of gastroptosis, which is usually part of a general enteroptosis. There is not time to discuss theories with regard to the causation of enteroptosis, but I would remind you that the appearance of dropped stomach is different from that of the definitely dilated stomach. The stomach is increased in a vertical diameter, whereas in the dilated stomach it is increased in the horizontal diameter. With a great deal of dropping an appearance very similar to hour-glass stomach is produced, the difference being that much of the trouble disappears in the recumbent posture, when emptying of the dropped stomach does not present the same difficulties. Other lines of treatment should be the attempt to restore tone to the stomach by abdominal message, by exercises, by drugs such as strychnine and arsenic, and by avoiding food which is liable to cause fermentation. Finally it may be necessary to employ some mechanical device such as the Curtis support combined with a belt. It is essential that in any such support the pressure shall not be directed simply backwards, but upwards and backwards, so as to push the stomach up.

An important form of dilated stomach is met with in infants who are the subjects of congenital hypertrophic stenosis, as it is called. It is probably not really congenital, however, but acquired in the first few weeks of life. And secondly, there is not true stenosis, but hypertrophy of the muscle of the pylorus.

It usually begins about the fourth week, and not later than the ninth. The forcible character of the vomiting is very characteristic, the child ejecting a quantity representing more than one feed a foot or more from the mouth, and sometimes through the nostrils as well. As the stomach dilates the frequency of the vomiting diminishes, while the amount increases. There is constipation and much wasting on inspection of the abdomen after food; vigorous contractions may be seen passing from left to right. It may be only during this time that you can feel the pylorus, which is so important a sign, but one which requires prolonged watching. Evidence of dilatation by auscultatory percussion can generally be obtained. The gastric contents tend to have an acidity below normal, but there is usually excess of rennin (Miller and Willcox).

I think the most plausible theory is that the condition is due to pancreatic insufficiency. The pancreas in the infant is always insufficient as far as starch digestion is concerned until the ninth month If there is delay in the development of the rest of the secretion of the pancreas pyloric spasm going on to hypertrophy is almost certain to occur. As soon as any acid of the gastric juice passes into the duodenum it should normally excite a secretion of pancreatic juice which will neutralise it. If pancreatic juice cannot be secreted the acid cannot be neutralised, and as long as there is acid in the duodenum the pyloric sphincter remains closed. So that if there is inadequate pancreatic secretion there must be pyloric spasm, and if this is prolonged it is highly probable that hypertrophy of the

muscle will follow. The unsatisfactory results given by operation also point in the same direction. If the contraction of the pylorus were the real prime factor in the disease gastro-jejunostomy ought to cure it, but we read that "after operation the infant's life hangs in the balance for several weeks," and on inquiring why the child is in this parlous state we find that after operation it is liable to intractable diarrhœa. That cannot be explained simply by pyloric obstruction, but if you throw into the intestines a quantity of food which they cannot digest because the pancreas is not secreting, it is easy to understand why the patient should suffer from intractable diarrhœa and be in such danger for several weeks. Another point is suggested: If you can keep the child alive until about the eighth or ninth month, the ultimate prospects are fairly good. I have gone into the theory of the condition because it has a distinct bearing on treatment. I am not prepared to recommend operation. A combination of repeated washing out of the stomach, with the administration of very small quantities of food which will not cause a residue, is the best treatment. At first only whey and raw meat juice should be given. The total amount of fluid given should not be more than a teaspoonful every twenty minutes. This should be slowly increased to two, three or four teaspoonfuls at longer intervals. Later, when milk is used, one to two grains of citrate of soda should be added to each ounce of milk to prevent curdling. According to Still, the stomach should be washed out with a solution of two grains of sodium carbonate to the ounce just before a feed twice daily for several weeks, and then once daily for some weeks longer. This treatment must be continued not only until vomiting ceases, but until the weight has begun to increase.

Lactic Acid Therapy.

In conclusion I should like to say a few words about lactic acid therapy. Perhaps it is less important for me to do so as there has been such a rapid subsidence in the lactic acid boom. The theory on which the treatment is based is that although it is impossible to give a sufficient quantity of antiseptic to render the alimentary canal sterile, we can greatly reduce the number of pathogenetic organisms there by giving a harmless organism which tends to inhibit the growth of the others. There is, of course, a considerable amount of truth in the statement; we can diminish the number of other bacteria in the intestine in this way. But that is no reason for administering soured milk when the evidence of intestinal intoxication is, to say the least, doubtful. I do not believe in the administration of any tablets professing to contain lactic acid ferments. The lactic acid producing bacillus is very fragile, and is hardly likely to stand compression into a tablet. Many such tablets will not yield the organism on culture, so that it is little use administering them to patients in the belief that they will get large quantities of lactic acid producing organisms therefrom. It is far safer to see that the milk is actually soured by the bacillus.

In the discussions on the subject of lactic acid therapy at the Royal Society of Medicine, and at the British Medical Association, the consensus of opinion seemed to be that benefit might be derived in three types of cases: first, those where the effect was mainly due to suggestion, in neurotic individuals, and those with imaginary maladies; secondly, those in which the treatment enabled the patient to take considerable quantities of nourishing and comparatively easily digested food. Allied to these is its usefulness in diabetes, where it enables the patient to take milk from which the sugar has been removed by fermentation. But even here caution is desirable in cases complicated by acidosis; and thirdly, cases in which we might expect a specific effect, because the microbe responsible for the intoxication or infection cannot flourish in an acid medium. The reaction of the fæces will be a guide. If the fæces are acid the treatment will probably not produce much effect. If the fæces are alkaline, but become acid in the course of a few hours, and evolve considerable gas in the tube, you will not have much result from lactic acid therapy. If, on the other hand, the fæces are alkaline and remain alkaline for twenty-four hours, and yield scarcely any gas to the tube, there is a reasonable prospect that the treatment will be beneficial. I should not advise it in conditions associated with hyperchlorhydria, for it is hardly reasonable to pour an extra quantity of lactic acid into a stomach which already has an over-dose of acid; besides, in hyperchlorhydria there is a fairly rapid destruction of the lactic acid producing organisms, for they flourish best in the absence of hydrochloric acid. And finally, the treatment seems to be contra-indicated in rheumatism. There is some clinical evidence that it has initiated or lighted up rheumatism again, which is interesting in view of the fact that the rheumatic diplococcus described by Poynton and Payne flourishes well in a lactic acid medium.

November 11th, 1912.

THE METHODS OF EXAMINATION IN OPHTHALMIC CASES.

By M. L. HEPBURN, M.D., B.S., F.R.C.S.,
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II.

SOME of the most valuable information in ophthalmic diagnosis can be gained by careful investigation of the iris, its reaction, colour, position, regularity or irregularity of the pupillary border, inequality, etc., and I advise you, whatever else you do, never to overlook the critical examination of this part of the eye, which is easily visible without the aid of magnifying lenses, and whose condition is the guide to so many different varieties of diseases of the eye.

Now the proper method of taking pupillary reaction does not seem to be generally understood, so that a detailed description will not be out of place. Arrange the patient opposite a window so that an equal amount of illumination is thrown on both eyes. Begin by examining the right one first. For this purpose the left eye must be entirely excluded from light by covering it with the right hand, then shade the right eye with the left hand so as to induce slight dilatation of the pupil. On removal of the left hand the right pupil ought to make a brisk contraction due to the direct action of light (= direct pupil reaction); next remove the right hand from the left eye, when a still further contraction should occur due to stimulation through the sensory visual tract on the left side (= consensual reaction). Repeat this performance on the left eye, and if both eyes act directly and consensually it shows that the reflex paths for the proper normal contraction of the pupil are intact in both eyes. Any variation in the pupil reaction is generally due to pathological causes. The colour, regularity, inequality, etc., is merely a matter of visual observation.

The first case I show you to-day is a typical one of acute iritis. You see there is:

(1) Ciliary injection.
(2) Discoloured iris.
(3) Fixed contracted pupil.
(4) Irregularity of pupillary border owing to adhesions.
(5) Normal depth of the anterior chamber.
(6) Normal tension.
(7) Cornea bright,
(8) Some pain.

You cannot too often contrast these signs and symptoms with those of acute glaucoma, of which I have no example here to-day, since they are almost immediately dealt with as soon as they present themselves at the hospital. They are:

(1) Slight ciliary injection.
(2) Fixed dilated pupil.
(3) Pupillary border regular, though perhaps oval or pear-shaped.
(4) Shallow anterior chamber.
(5) Cornea steamy.
(6) Tension raised.
(7) Pain in the head localised to the side of the affected eye.
(8) Possibly vomiting.

I have, however, a case of chronic glaucoma in a man, æt. 40 years, which I now show you. For two years he has been slowly losing the sight of the left eye, and as he did not seek advice the sight has gradually been lost so that he is now unable even to perceive light. His right eye is also the subject of glaucoma though the vision is not yet decidedly affected. You will observe there are none of the signs I have just enumerated for the detection of glaucoma, and the only evidence of any importance externally is the inability of the iris to maintain its contraction when light is thrown into the eye by oblique illumination; but this is an accompaniment of affections of any part of the sensory visual tract (*e.g.* optic atrophy), so that it only serves as a warning for making as careful an examination as possible. The fundus shows that the discs are both cupped, which is the only other sign of the presence of glaucoma. It is at once obvious to you that these cases are difficult to recognise, and in addition to the signs given above the only history to be elicited is one of temporary mists and haloes accompanied by some pain due to rise of tension; these, however, pass off in a short time, so that at the time of examination they are no longer present, and deterioration of visual acuity is all that remains of these periodical attacks of active glaucoma.

Here is a case of fixed dilated pupil on the right side with a blind injured eye on the other due to a blow many years ago. There are no signs of acute glaucoma in this case, but the pupil neither acts to light nor accommodation. This indicates

either some defect of the sensory part of the reflex arc in either eye or paralysis of the third nerve. If optic atrophy were present there would be no action of the pupil, since there is no possibility of stimulation of the third nerve through the sensory tract in either eye, and thus the pupil might remain fully dilated; but there is no pallor of the optic disc and the vision is perfectly normal. Therefore the only conclusion to be drawn in order to account for the fixed dilated pupil is the instillation of a mydriatic; this the patient denies, though I am afraid it has occurred without his knowledge through some error in the application of remedies.

The next case is one of orbital growth producing proptosis of the right eye which has existed for five years. Now although in this particular instance the proptosis is unmistakable sometimes its presence is a matter of doubt. The method of examination is to place the patient in a chair and stand behind her; with the first finger of each hand placed upon the upper orbital margin raise the upper lids and arrange a point of view so that the front of the cornea on the sound side can just be seen in a line with the upper margin of the orbit; then compare the two sides, and you will see that in this case the right eye is distinctly more prominent than the left and is pushed forwards and slightly downwards.

Whenever proptosis is present it always points to some affection of the orbital cavity (apart, of course, from exophthalmic goitre), but in addition to this sign three others of equal importance must be looked for; these are chemosis (or œdema of the conjunctiva), limitation of movement, and the condition of the fundus, especially of the optic nerve. There must be such complete association of these four signs in the mind that any one of them will suggest the possibility of the presence of any or all the other three. In the case I am showing you here the chemosis is only slight, indicating very little obstruction in the orbital vascular system, which is an important piece of evidence taken in conjunction with the very marked proptosis. The limitation of movement is mostly upward, though abduction and adduction are not complete, and the downward movement is unimpaired. The fundus is perfectly normal and the vision is $\frac{6}{9}$. Now we are in a position to diagnose our case. The eye being pushed downwards and forwards with limitation of movement upwards suggests pressure on the globe by some swelling which is situated above and has extended downwards slightly behind the eyeball. The absence of chemosis and the fact that the optic nerve is normal shows that the latter as well as the vascular system has not been involved, although the swelling has existed for so long a time, and the strong probability is, therefore, that whatever the growth is it is not malignant, but is probably an innocent tumour growing from the upper wall of the orbit. The swelling felt through the skin of the upper lid is in favour of an orbital exostosis and no operation is thought advisable. It is always well to bear in mind the anatomical relations of the orbit and the proximity of the various sinuses in the neighbourhood, and often the direction of the proptosis is indicative of the particular sinus involved.

I now want to show you three cases in the dark room in order to illustrate a rough but extremely efficient method of ophthalmoscopic diagnosis. Examine each one in the same way, namely, by projecting light into the eye from the large concave mirror of the ophthalmoscope, which must be held up to the surgeon's eye at a distance of about a metre. Under these circumstances we observe a uniform bright red reflex illuminating the pupillary area from the fundus in whatever direction we throw the light. In the first case we see a stationary opacity showing up as a dark area against the red reflex, which is seen to be normal round the edge. The only places for a stationary opacity to occur is in the cornea or the lens, and since we can exclude the former by other methods of examination we know that the lens is the part affected. We can, therefore, diagnose some variety of cataract.

The second case is one of detachment of the retina. Here there is no obstruction to the red reflex but it is not uniform in every position; in the upper part it looks normal, but below there is a greyish-white reflex instead of the ordinary red colour, indicating that the fundus is not normal over this area; it may require other methods of diagnosis to determine the exact nature of the condition, but the fact that an abnormality exists in this situation is established.

The third case shows in various places a greyish reflex sprinkled with black spots. Now masses of pigment are always associated with inflammation of the choroid, and although it may be impossible without considerable experience in the use of the ophthalmoscope to state the exact form of choroiditis present, yet we are in a position to say that this particular form of inflammation has occurred.

This method of ophthalmoscopic examination, which is easily acquired, is extremely valuable and rapid; it is, of course, impossible for anyone to form an opinion as to detail, but at any rate it indicates the part of the eye which is affected, thus locating the lesion, which is often the most difficult part of ophthalmoscopic diagnosis to the general practitioner.

November 11th, 1912.

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A CLINICAL LECTURE

ON

THE SIGNIFICANCE AND TREATMENT OF SUGAR IN THE URINE DURING PREGNANCY.

Delivered at St. Bartholomew's Hospital.

By HERBERT WILLIAMSON, M.B., B.C., F.R.C.P.,

Assistant Obstetric Physician at the Hospital.

GENTLEMEN,—I want to take for our consideration this morning a case which has recently been under my care in the lying-in ward here. It is a case of pregnancy complicated by the presence of sugar in the urine.

Let me say first that the significance of the presence of sugar in the urine of pregnant women depends to a great extent upon the nature and quantity of the sugar. Its presence may be merely a transient phenomenon, of no clinical importance, or it may be a sign of gravest danger to both mother and child. It is clear, therefore, that in dealing with any given case we must in the first instance endeavour to place it in the particular group to which it belongs.

Remember that the occurrence of traces of sugar in the urine during pregnancy is a common phenomenon. If you take a large series of women who are pregnant and examine their urine a day or two before delivery, employing delicate tests, you will find in it not uncommonly traces of both glucose and lactose. This indicates an alteration in metabolism, of which I shall have to speak more in detail in a few moments. You may, however, take it as a working rule that any case in which sugar is present in sufficient quantity to give a reduction by Fehling's test calls for a careful investigation, but if, on the other hand, the quantity is not sufficient to be detected by that test, the case need cause no anxiety.

We will suppose you have examined the urine of a pregnant woman by this test and you find it contains sugar; your next step in the investigation is to ascertain what kind of sugar is present. There are only two sugars we need consider, for all other forms are very rare; the two of importance are lactose and glucose. Now, the significance of the presence of these two sugars is very different, therefore our next investigation must be to ascertain whether we have to deal with lactosuria or glycosuria. Both lactose and glucose reduce Fehling's reagent, so that it is necessary to employ some other test to differentiate between them. There is one test, very easily applied, which for clinical purposes may be relied upon, and that is the fermentation test. Glucose is fermented by yeast, lactose is not fermented by yeast, but when you employ this test you should first boil the urine to render it sterile.

When you have ascertained, therefore, that the patient's urine reduces Fehling's reagent, you next try the fermentation test. If the sugar undergoes fermentation by means of yeast you are dealing with glucose, and this is a serious matter, and if it does not undergo fermentation you are dealing with lactose; but remember that occasionally both lactose and glucose may be present at the same time. There are two other tests which may be applied, but they are more difficult, and, generally speaking, are only applicable where there is a laboratory. One is by the preparation of the osazone crystals, for lactosazone crystals differ from glucosazone crystals; the other test is by the polarimeter, and depends upon the fact that whilst glucose and lactose are both dextro-rotatory, the angle of rotation differs in the two solutions. These two tests are luxuries not always available, and for practical purposes we may say that if the sugar is fermented by yeast it is glucose, if it is not it is lactose.

Let us take first a case in which the urine reduces Fehling's solution but is not fermented by yeast. This means we are dealing with lactose, and before talking about the graver subject of glycosuria I will say a word about lactosuria.

A question naturally arises here. How is lactose formed? A woman is pregnant, and has no lactose in her urine before the baby is born; two or three days after the birth of the child she excretes lactose in considerable quantities. How is this brought about? Where does the sugar come from? There have been some interesting experiments made on this subject recently. First of all, let us ask what happens to an animal if you amputate its breasts and then allow it to become pregnant? Will that animal form lactose, or not? You find a very interesting result follows the operation. The breasts of a bitch were amputated, and afterwards that bitch became pregnant and gave birth to puppies. No lactose appeared in the urine, but on the day when lactation usually commences the urine contained a quantity of glucose. From this experiment it appears that one function of the breast is to pick glucose out of the blood and convert it into lactose. That is a very important observation, and helps us to understand the significance of lactose in the urine of the pregnant woman. The presence of lactose simply indicates premature mammary activity; for some reason, before the birth of the baby the breasts begin to function and to form lactose. The lactose is reabsorbed from the milk-ducts, passes into the blood-stream and is excreted in the urine. I have been trying to find a pregnant woman who has had both her breasts removed but I have not succeeded, though I have a patient who became pregnant after the amputation of one breast, and her urine after delivery was found to contain lactose but no glucose. It would be extremely interesting to see what would happen in a woman who was pregnant after both her breasts had been removed; one would expect, from the experiments upon animals, that her urine would then contain considerable quantities of glucose.

In what proportion of pregnant women are we likely to find lactose in the urine? Out of 100 consecutive pregnant women who presented themselves in the out-patient department to apply for maternity letters we found that in three cases only could lactose bedetected by Fehling's test, but by using more delicate methods minute quantities may be detected in 20 to 30 per cent. of the patients. It is therefore not very common for these women to have a sufficient quantity of sugar in the urine to reduce Fehling's solution before delivery. Its presence probably means that the breasts commence to function and to form lactose a little too soon, and in the light of this interpretation it has no clinical significance.

Now let us consider the urine after delivery.

The presence of lactose in the urine after delivery is extremely common ; almost all women have it about the third day, when the breasts become engorged, and the more distended the breasts the larger is the quantity of lactose found in the urine. When the function of lactation becomes well established and the woman is comfortably nursing her baby, the breasts do not become distended and there is no lactose in the urine, but if for any reason the breasts again become distended and engorged, lactose re-appears in the urine. Again, we find almost invariably a considerable amount of lactose just at the time of weaning. You see that often in hospital practice : a nursing mother has to have some operation performed and the baby is weaned ; the urine is examined and found to contain a considerable quantity of lactose.

To sum up, we may say that a trace of lactose is common during the latter weeks of pregnancy, and indicates merely premature mammary activity but has no further significance clinically ; that after delivery whenever the breasts become engorged lactose is present in the urine, and when the distension of the breasts is relieved the lactose disappears.

We will now put aside those cases in which we find lactose, and turn to the cases in which we find glucose in the urine. The subject was investigated carefully by the late Dr. Matthews Duncan in 1882, and he wrote a gloomy paper in which he reported that of sixteen patients suffering from glycosuria during pregnancy eleven were dead within two years of delivery. From the date of that paper until recently the presence of even small quantities of glucose in the urine was regarded as a matter of the gravest significance, and a bad prognosis was given. It is clear, however, that all cases of glycosuria do not belong to the same category, and we must now endeavour to distinguish between the different groups, and to see what tests we can apply to enable us to put a given case into the group to which it belongs.

As I have said, traces of glucose not discoverable by Fehling's test are present in about 20 per cent. of pregnant women in the last weeks of their pregnancy, but glucose in sufficient quantity to be readily detected by Fehling's test is rare. Out of 500 patients in the maternity ward here glucose has been discovered in the urine of four only, and to one of these I wish to draw your attention this morning.

Cases of glycosuria fall into three groups : First of all there is what we may call *alimentary glycosuria*—that is, glycosuria dependent upon the ingestion of certain foods. Secondly, there is *transient glycosuria of pregnancy*, cases in which glucose is present in the urine in pregnancy and persists in spite of dieting, but after delivery disappears completely, and does not return unless the woman becomes pregnant again. Thirdly, there is true diabetes. In these three groups the prognosis is very different both as regards the mother and the child. Let us, then, consider these three groups a little further.

I will first give you a very brief account of the case of which I have already spoken, because it is a very interesting one. The patient, a woman, æt. 21 years, was pregnant for the first time. When her urine was examined in the eight month of pregnancy it was found to contain albumen, and in addition 40 gr. of sugar to the ounce. She was passing a large quantity of urine, and was suffering considerably from thirst and from severe pruritis, particularly of the vulva. She had not a voracious appetite. Further investigation of the urine showed that the whole of the sugar was glucose, and that the urine contained both acetone and diacetic acid. We took the patient into hospital, and for the first few days kept her under observation without making any alteration in her diet. During those days she passed 18 gr. of sugar to the ounce. She was admitted on September 18th last year. On the 24th we stopped all sugar, and by this means alone the amount excreted in the urine fell from 40 gr. to 13 gr. to the ounce. On September 28th we excluded from the diet potatoes, and that again markedly decreased the amount of sugar passed, for it fell from 13 gr. to 8 gr. to the ounce. On September 30th she was placed upon a strict diabetic diet, and then the sugar fell to a mere trace. You will say that was a case of alimentary glycosuria, and so I thought, but the sequel shows it was not. On October 1st she went into labour, and we delivered her of a living child. On the third day after delivery we found sugar again in the urine, but this time it was proved to be lactose, and obviously was connected with commencing mammary activity, so we felt happy about her, but we kept her under

observation, gradually increasing the amount of carbohydrate in the diet. Before discharging her from hospital we gave her two test-meals and then took observations. On the first occasion we gave her 50 grm. of glucose and made careful examinations of the urine at intervals during the next twenty-four hours, but there was no trace of sugar. A few days afterwards we gave 100 grm. of glucose by the mouth, and that was followed by the appearance of a small quantity of sugar in the urine, but it did not persist, and we concluded that her carbohydrate tolerance was re-established. I heard nothing more about her for some months; then her doctor wrote to say that she was again pregnant and was passing sugar in her urine. She was again admitted, and when I examined her I found she was two months pregnant. The urine contained 21 gr. of sugar to the ounce, and this time the most careful dieting failed to diminish the quantity. After consultation with Dr. Garrod I emptied the uterus and the sugar immediately fell to 10 gr. to the ounce, but it never disappeared. She went out, and since that time she has been gradually going downhill; she is suffering from true diabetes, and I do not think she will live very long.

I want next to say a few words about the group of cases I mentioned to you first, namely, alimentary glycosuria. I think the best example I know is a case recorded by Whitridge Williams. The patient was a healthy woman, æt. 24 years. In her first pregnancy glucose was present in the urine during the last two months, but disappeared immediately after delivery. She became pregnant a second time, and during her second pregnancy she again had glucose in the urine; after delivery occurred it disappeared. "In her third pregnancy sugar made its appearance in the seventh month, and persisted in varying amounts until the birth of the child. She presented no symptoms, but she passed 60 oz. of urine in twenty-four hours, and the urine contained 4 per cent. of glucose Its quantity remained unchanged, whether she was upon ordinary diet or upon carbohydrate-free diet On discussing the matter with the patient I found she had suffered from constipation for years, and she had been making and taking a mixture of senna and prunes; it was prepared by stewing a pound of prunes and a handful of senna-leaves in a quart of water, to which one pound of sugar was added, and evaporated down to a thick syrup. Each night she took half a dozen prunes with a considerable quantity of the syrup. I thought the glycosuria might be due to the fact that she was unable to assimilate this extra amount of sugar, and that was found to be correct. On stopping the taking of this mixture all traces of sugar disappeared from the urine, and upon retaking the mixture sugar reappeared in the urine. She was intolerant of sugar administered in this form, but she was able to deal with sugar administered in the ordinary way. Her glycosuria was dependent on the fact that she was taking more carbohydrate than she could deal with."

Interesting observations have been made, which prove that a woman during pregnancy is not able to deal with the same amount of carbohydrate as in the non-gravid state. If you take a number of pregnant women and an equal number of non-gravid women and administer to them 100 gr. of glucose you will find glycosuria in 70 per cent. of the pregnant but in only 16 per cent. of the non-gravid, and if you employ 200 gr. of glucose you will find glycosuria in 100 per cent. of the pregnant but in only 29 per cent. of the non-gravid. That is a very striking experiment, showing clearly that pregnant women are notable to assimilate the same amount of sugar as the non-gravid. This experiment, to my mind, affords an explanation of many of these cases of alimentary glycosuria, the presence of glucose depends upon some alteration in the metabolism of the pregnant woman, an alteration which unfits her to deal with the same amount of carbohydrate as she can deal with in the non-pregnant state. It is obvious, then, that when you have discovered that a woman's urine contains glucose, you must ask yourself this question—Is this a case of alimentary glycosuria? To answer it you must go carefully into the question of diet and see if the sugar disappears with a carbohydrate-free diet. If it does disappear the case is probably one of alimentary glycosuria, but you must remember that you cannot be certain about this, because in the case I have just related to you what appeared to be an alimentary glycosuria was in reality the commencement of true diabetes.

Now a few words about the cases we call *transient glycosuria of pregnancy*. There are some pregnant women in whose urine glucose is found,

and the quantity of that substance is not materially affected by diet, yet when the pregnancy is over the glucose disappears and there is no recurrence, even in the course of years, unless the woman again becomes pregnant. As I shall tell you in a moment, it is almost impossible during pregnancy to differentiate such cases from those of true diabetes. What is the explanation? I think it may possibly be found in the condition of the pituitary body. We know two things about the pituitary body. We know, in the first place, that it exercises a profound influence upon carbohydrate metabolism, and in cases of acromegaly when the pituitary body is affected the patients often pass sugar in the urine. We know, in the second place, that it undergoes a very marked series of changes during pregnancy; amongst these changes is hypertrophy. There was an interesting case mentioned in the 'British Medical Journal' a few weeks ago, that of a woman who, when she became pregnant, developed symptoms of acromegaly but recovered when the pregnancy terminated. It is probable that these cases of transient glycosuria depend upon some change in the pituitary body. This is not proved, but it is the most reasonable explanation yet offered.

Now we must turn our attention to the subject of true diabetes. In the first place it used to be thought that diabetes always caused sterility, and that a diabetic woman never became pregnant. That is, however, an erroneous idea. It is perfectly true that in some cases diabetes is associated with changes in the ovaries, that the formation of ova ceases and the ovaries become converted into fibrous tissue, but this is not always the case, and it is not uncommon for a woman who is the subject of true diabetes to become pregnant. Diabetes may commence during pregnancy, as it almost certainly did in the case I have related to you, but it may equally well have existed for some time previously and be first discovered when the routine examination of the urine is made during pregnancy, for we know that people sometimes first learn that they have diabetes when they go to be examined for life assurance.

Let us consider what is likely to happen if a diabetic woman becomes pregnant. Has diabetes any effect upon the pregnancy? Yes, it has a marked influence. In diabetic women miscarriages are common, and if the patient does not miscarry the child is likely to die during the last week or two before delivery. Next, there is often a very remarkable complication, namely hydramnios, and you may find sugar present in large quantities in the liquor amnii. I read an amusing description a short time ago of a case reported from America, in which it was stated that the child was coated with sugar when it was born. I confess I have never seen such a condition as that, but I have on one occasion found a considerable quantity of sugar in the liquor amnii. Another curious fact is that in some cases of diabetes you have excessive development in the child, so that labour may be obstructed and perforation or embryotomy has to be performed. The question of excessive development is interesting, because you know it has been suggested that in the case of women who habitually have large children, the size of the child may be reduced by cutting off the carbohydrates from the diet of the mother during pregnancy.

Let us now consider what effects pregnancy produces on the course of diabetes. In the first place remember that marked exacerbation of the symptoms does not usually occur until the later months, but there are many exceptions to this rule. Usually about the sixth or seventh month the symptoms become aggravated; the patient develops excessive thirst, has often a voracious appetite, sometimes passes large quantities of urine, and also suffers from severe pruritus vulvæ. At this stage a certain number, and a not very inconsiderable number, pass into coma and die, but you must remember that coma more commonly supervenes shortly after delivery. An explanation has been offered to account for this last fact. The coma which these diabetics develop is one manifestation of acidosis, and is associated with the presence of acetone and diacetic acid in the urine. Acidosis is markedly increased by muscular effort, and there is no doubt that the muscular exertion of labour and the contractions of the uterus are the immediate cause of the coma. This is important to remember, because I shall mention in a moment a principle of treatment which we adopt in these cases to help to tide over the difficulty.

Now as to the prognosis. The prognosis in these cases is bad. Twenty-five per cent. of cases of true diabetes die within twenty-four hours of delivery; and by the end of two years another

25 per cent. of them are dead. So that 50 per cent. of patients suffering from true diabetes will be dead at the end of two years from delivery. So you see the prognosis is often extremely grave. Of course there are exceptions. You may meet with women who are diabetic and yet go through repeated pregnancies without very much disturbance. I have never seen such a case personally, but Lop has given us an account of a woman who passed through six pregnancies with very little disturbance after the diagnosis of true diabetes had been definitely established. That is a very exceptional case, but the fact that such a thing has happened should make us very careful about giving a prognosis in any given case. Remember, then, that the prognosis is very grave, but you may meet with cases in which pregnancy does not seem to do the patient much harm.

On the question of diagnosis I have not much to say. If you find in the urine of a pregnant patient sufficient sugar to reduce Fehling's solution, find out whether it is glucose, or whether it is lactose. If it is lactose there is no need to worry about it. If it is glucose you must determine to which of the three groups I have mentioned the case belongs. Is it alimentary glycosuria? Is it transient glycosuria? Is it true diabetes? First you will put the patient on a strict diet, and if the sugar disappears under that strict diet, probably, but not certainly, you are dealing with merely alimentary glycosuria. You cannot be certain, however, until after delivery. To distinguish between transient glycosuria of pregnancy and true diabetes is a very difficult matter, so difficult, in fact, that it is usually impossible until after delivery has taken place. Generally speaking, one may say (1) that if you find sugar present in the early months of pregnancy the case is probably one of true diabetes, and not transient glycosuria, for the latter condition is seldom found before the sixth month; (2) if you find that the sugar persists in considerable quantity in spite of diet, it is probably true diabetes and not transient glycosuria; (3) if you find there is a marked increase in the appetite and much thirst, it is probably diabetes and not transient glycosuria; and (4) if you find in the urine the presence of acetone or diacetic acid, it is probably true diabetes and not transient glycosuria. You cannot, however, give a positive opinion until labour is over, and you see whether the sugar disappears or persists.

Now with regard to treatment. If any of the grave symptoms I have mentioned are present, if the sugar persists to the extent of 3 or 4 per cent. in spite of dieting, if the patient is suffering markedly from thirst and from increase of appetite, or if you find acetone or diacetic acid in the urine, there is no question that the pregnancy should be terminated. The risk of allowing the pregnancy to continue is great, and the life of the child is so problematical that it is not right to endanger the life of the mother. There is a practical point with regard to the termination of the pregnancy to which I wish especially to draw your attention. It is that you should never administer chloroform in these cases; acidosis is a marked feature of chloroform poisoning, and it is dangerous to increase the intensity of the acidosis already existing. If any anæsthetic is required ether should be given. The method of induction to be adopted depends on the stage at which the pregnancy has arrived. If the sixth month has been reached, the best way is to introduce bougies between the uterine walls and the membranes. In the earlier months of pregnancy the better method is to dilate the cervix by means of a laminaria tent. If, as in my case, you can reduce the amount of sugar, if the general health is not suffering, if there is no excessive thirst or voracious appetite, you should allow the pregnancy to continue, because in such a case you have a very good chance of getting a living baby. But if at any time during pregnancy the patient develops these grave symptoms, you should not hesitate to end the pregnancy. It is important to bear in mind the fact that acidosis may be induced by the continued muscular contractions of the uterus. The reason a diabetic woman goes into coma so often after delivery is because these acid bodies are elaborated through the muscular exertion, and therefore at the time of delivery you should take precautions against this danger. If you estimate the alkalinity of the blood in these women you find it is much diminished; it is obvious, therefore, that if we are to ward off acidosis it is necessary to increase the alkalinity of the blood, and the best way of doing this is by intra-venous infusion with sodium acetate solution. At the commencement of labour we therefore run in two pints of normal saline solution, to which one drachm

of sodium acetate to the pint has been added. This has a marked effect in increasing the alkalinity of the blood, and is of real practical importance, for knowing that your diabetic patient is liable to coma, after delivery and during labour it is wise to infuse intra-venously a couple of pints of saline solution to which acetate of sodium has been added.

The whole question of glycosuria in pregnancy is a difficult and complicated one, and as we have been investigating it during the last eighteen months I thought I would put before you the results at which we have arrived. I have endeavoured to show you that it is not sufficient to rest content with a diagnosis of glycosuria, but that when you have made that diagnosis there are still three distinct conditions to be differentiated: alimentary glycosuria, transient glycosuria of pregnancy, and true diabetes. The differentiation between these three conditions is not simple, and the case I have read you shows how extremely difficult it may be, for in this patient the sugar disappeared and did not return when we gave first 50 grm. of glucose and later 100 grm. by the mouth, and yet the patient proved to have true diabetes.

November 18th, 1912.

Mr. A. H. TUBBY has published his second edition of 'Deformities, including Diseases of the Bones and Joints,' through Messrs. Macmillan & Co. The work appears in two volumes. The author has not failed to appreciate the immense progress that has been made in orthopædic surgery, and has not scrupled to pass over older methods of treatment in those cases in which new methods based on clearer conceptions of pathogenesis and recent advances in technique have come into vogue. This difficult task the author has accomplished in a masterly manner, and his work enables one to say that at last we have a recent British work dealing fully with orthopædic surgery, and so successfully achieving this difficult enterprise that it becomes the classical work on the subject. In the last edition Mr. Tubby adopted the method of regional classification in the grouping and arranging of the various subjects dealt with, but in his new edition the author has, in our opinion, adopted the wise course of writing his book in such a manner that the questions dealt with are put forward not only in regard to treatment but also in regard to ætiology and pathology, these last two considerations being fully set out. We heartily recommend this remarkable book to the attention of our readers.

THREE LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
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LECTURE I.

EARLY HISTORY: B.C. 450–A.D. 1854.

AMONG primitive people every able-bodied man takes up arms in time of danger to protect his own tribe or village, and it is only as States become more civilised that an army arises as an organised disciplined force, acting in unison and in obedience to the command of officers. Such forces were raised in Egypt in the days of King Rameses II, the Pharaoh of the Oppression, about 1322 B.C., and by such troops he conquered less well-equipped countries, and is said to have penetrated into Asia as far as India. But in the art of warfare, as in other matters, ancient Egypt became, first non-progressive, and then decadent. A little later in the world's history Persia and Greece, and then Rome, each had their splendid armies, organised in times of peace and overwhelming in time of war. But military organisation perished for a time with the invasion of the northern barbarians into the Roman Empire, when tactics and strategy disappeared and gave place to armed mobs, whose success was chiefly due to numerical strength or individual valour.

The Turkish Janissary force, organised in 1362 A.D., was the first standing army in Europe, and the superiority of such an army over those levied only in war time, and consisting of untrained men was so obvious that other countries adopted the idea. In England the national militia of Saxon days gave place to armies which were raised by the barons. Cromwell established, it is true, a standing army, but it was very unpopular, and Charles II was forced to disband all his trained soldiers, with the exception of a body-guard and the Household Brigade of 5000 men. In succeeding reigns, when war broke out an army was levied, but when hostilities ceased it was disbanded, always with the ex-

* Gresham Lectures delivered in October, 1912.

ception of a small nucleus, which increased in size as time went on. Corps of artillery and engineers were added, and in the Peninsula war the army was organised into divisions, and the commissariat and transport were brought to a high standard. But again, at the end of the war, the forces were reduced from 246,000 to 72,000, and every department of military organisation was left in a condition of distressing inefficiency. The result of such a state of things was the chaos from which every branch of the British army suffered at the outbreak of the Crimean war in 1854.

To trace the history of organised military art is to study the origin and growth of the Army Medical Service. Among barbarous people the wounded are mostly left to perish on the battlefield as useless encumbrances, and the conquerors rob and murder them. But as civilisation advances humane methods of warfare take the place of such brutalities, and it becomes incumbent upon the commander of an army, not only to give the utmost care to his own wounded, but, in common humanity, to take pity upon the helpless victims of warfare on the enemy's side. Thus the need of the army surgeon and proper methods for the transport of the sick and wounded arise.

The earliest military surgeons of whom we have any authentic reports are those who were attached to the Greek armies. Thus in the Persian wars, about 450 B.C., a physician of the name of Onasilos, with two of his pupils, volunteered to follow the army to attend the wounded, and, according to the inscription which records this service, they were rewarded by money or lands. There are other records of surgeons and physicians of Greece who acted as medical attendants on the army, but there appears to have been no provision made to convey the wounded from off the battlefield. These had to depend upon their comrades, who carried them on their backs, and the story is recorded of one such bearer who was caught trying to bury his burden while yet alive. For this Xenophon ordered him to be publicly whipped, and when the soldier eventually accused his general of undue severity, public opinion in the army merely decided that the punishment had not been heavy enough. It was the ideal of Xenophon to employ the best surgeons for the care, not only of his own wounded, but of those of the enemy. When the Greeks fought at Sparta it is recorded that the people of Lacedæmon opened their houses for the reception of the wounded, and tended them in a fashion worthy of great citizens. It was Greek surgeons who served with the Roman armies in the beginning of our present era, probably because the Roman citizens considered medicine an art beneath their dignity, and, therefore, employed foreigners to minister to them in sickness. But each citizen soldier had to carry with him into battle, as in modern times, bandages with which he knew how to dress a wound. After a battle the wounded were received into the houses of the rich, and it is reported that they were conveyed in vehicles, which may possibly have been early ambulance waggons. Army surgeons of various ranks were attached to each cohort of Roman soldiers; they wore the dress and arms of the legionary, and ranked with the standard bearers and trumpeters. Besides the surgeons there were eight to ten strong men attached to each troop of about 200 to 400 men, who had to ride behind the fighting line to pick up the wounded. To enable them to do this they were fitted out with two stirrups on the left side, water flasks, and, possibly, bandages. They received a piece of gold for each man they rescued. Homer, Livy, and Tacitus all tell us stories of rough surgery and bandaging, and of men and women skilled in remedies to check bleeding and alleviate pain.

Passing to mediæval times, we hear that the ancient warriors of Germany were attended by their wives and daughters after a battle, and those skilled in leech-craft received handsome rewards for similar service. But these ministering angels did not venture on to the battle-field. Those who were unable to reach home presumably received no treatment, though it is probable that a certain number of adventurous healers may have followed the army for the sake of attending to the nobles and picking up what fees they could from grateful patients.

We know nothing of the healers who attended our warlike kings prior to the conquest, but in Domesday Book there are recorded the names of two medical attendants who landed with William the Conqueror, and both were royally rewarded by gifts of land, so we may conclude that their services were appreciated.

The early Crusaders appear to have been unaccompanied, either by priests or by doctors. But

Carte, the English historian, tells the story of the wound Prince Edward, later Edward I, received at Jaffa, and here an English surgeon takes a prominent part, more prominent than the wife Eleanor, who is popularly supposed to have sucked the poison from the wound.* "The assassin," he tells us, "drawing a poisoned dagger, attempted to stab the Prince in the belly, but the Prince, endeavouring to parry it with his arm, received there a deep wound, and, striking at the villain's heels with his foot, seized the dagger and plunged it in his heart, though in wresting it from him he gave himself a wound in the forehead. The wound in his arm appearing dangerous and likely to gangrene, the Prince thought fit the next day to make a will; but the bad flesh being cut away by an English surgeon, it was healed in little more than a fortnight." This experience must have taught Edward the value of medical service in warfare, and in 1299 this same monarch was accompanied by seven doctors when he invaded Scotland. There is, however, no evidence that the bulk of the army received any attention from surgeons or nurses when they were wounded.

There is no record of any nation inaugurating any properly constituted medical service for the soldiers, or any field hospitals or ambulance corps, till the reign of Queen Isabella I of Castile, known as Isabella the Catholic. To us this gifted woman is identified chiefly as a patroness of Columbus, and, alas! with the horrors of the Inquisition, which was established in her reign, but it must be remembered that her motives, even then, were pure, and her zeal for her religion undoubtedly blinded her to the wickedness of the means employed. But her religion also taught her mercy and charity for her subjects, and her pity extended to the vanquished in war. The historian, Hernando del Pulgar, says of her: "For the care of the sick and wounded the Queen sent always to the camp six large tents and their furniture, together with physicians, surgeons, medicines and attendants, and commanded that they should charge nothing, for she would pay all. These tents were called the Queen's Hospital." The Spanish army in 1487, was accompanied by 400 waggons containing the Queen's hospital, and these waggons were named "ambulancias."* Peter Martyr, writing at the siege of Grenada, says: "Four huge hospital tents, the careful provision of queenly piety, are a sight worth seeing. They are intended, not only for the wounded, but for those labouring under any disease. The physicians, apothecaries, surgeons and other attendants are numerous, and order, diligence and the supply of all things needful complete. . . . Every sickness and casualty is met and provided for by the royal bounty, except where Nature's appointed day is at hand." The Queen herself visited her stricken soldiers, saying: "Let me go to them, for they have no mothers here, and it will soothe them in their pain and weakness to find that they are not uncared for." Unhappily the humane methods introduced by this great woman were not continued in later wars.

The King and great seigneurs of France took their body surgeons with them on a campaign, whilst the soldiers had to content themselves with quacks and barber surgeons who followed the flag. Among the latter was Ambroise Paré, who started his military life in the expedition to Turin in 1536. He had no recognised position in the army; he was paid by the job in money or in kind, and attached himself to various great men, till he was made surgeon-in-ordinary to the King of France. There were no field hospitals and no nurses; the wounded had to get along as best they could unless the inhabitants of the neighbourhood took pity on them and gave them shelter. But some at least of Paré's patients were more fortunate, as we learn from the following story quoted from the pages of 'Journeys in Diverse Places,' translated by Mr. Stephen Paget:

"One of the servants of the Captain Ensign of the Company of M. de Rohan went with others to enter a church where the peasants had retreated, thinking to get victuals by love or by force; but he got the worst of it, as they all did, and came back with seven sword wounds on the head, the least of which penetrated to the inner table of the skull; and he had four other wounds upon the arms, and one on the right shoulder, which cut more

*"Or her who knew that Love can vanquish Death,
Who kneeling, with one arm about her King,
Drew forth the poison with her balmy breath,
Sweet as new buds in Spring."
(Tennyson's 'Dream of Fair Women.')

* This is probably the first time we meet with the word "ambulance," which, although mentioned in English literature as early as 1819, did not come into general English use until the Crimean war.

than half of the blade-bone. He was brought back to his master's lodgings, who, seeing him so mutilated, and not hoping he could be cured, made him a grave, and would have cast him therein, saying that otherwise the peasants would torture and kill him. I, in pity, told him the man might still be cured if he were well dressed. Diverse gentlemen of the company prayed he would take him along with the baggage, since I was willing to dress him, to which he agreed, and after I had got the man ready he was put in a cart, on a bed, well covered and well arranged, drawn by a horse. I did him the office of physician, apothecary, surgeon and cook. I dressed him to the end of his case, and God healed him, insomuch that all the three companies marvelled at his cure. The men of arms of the company of M. de Rohan gave me each a crown and the archers half a crown."

This, then, was the condition of things in the middle ages, with no organised army medical service; volunteers from the medical world, generally men of no recognised position or standing, followed the troops and assisted them with more or less skill. Now that better ways have prevailed it is unnecessary to touch upon the fate of those who procured no assistance or were abandoned for want of any transport service. It is astonishing how light-heartedly brave men went into wars with the likelihood of such a fate awaiting them. As methods of warfare improved the means of mutual destruction, succour for the wounded happily also came to be regarded as of urgent importance.

A sense of responsibility towards the afflicted is the invariable accompaniment of a higher civilisation. People would not tolerate the haphazard methods of more barbarous days, and great generals awoke to the fact that they must provide effective assistance for the casualties of war.

But this did not come quickly, and even so far-seeing a general as Napoleon I paid such insufficient attention to his medical corps as no modern army would tolerate. Abundant evidence of this is furnished in the accounts left by the army surgeons engaged in his campaigns. Thus Dr. Percy wrote in 1807: "During the siege of Dantzic, where we had 1600 wounded and 2000 sick, we had not a single straw mattress, no basins, no nurses; in fact, there was nothing." "One day, seeing more than eighty carriages full of sick and wounded soldiers arriving at a hospital in which there were neither nurses or attendants, Percy, who was about to leave, delayed his departure in order that he might attend to them. For five days the greater part of them had not left the cart which had served for bed and transport." "With shame be it written, there was nothing for these poor people to lie upon, not a mattress, nor even straw . . . no linen, no candles, no food. There were no means to change the dressings of a wound, nor even to warm these unfortunate wretches. We waited until nine o'clock at night, hoping that assistance would arrive, but we saw nobody except a person who came to count the victims of this most culpable improvidence, this most barbarous carelessness."

"Six days after the battle of Eylau (1807), the wounded who had betaken themselves or had been carried to Thorn, a distance of five leagues, were in as deplorable a state as they were during the first hour after the battle. Nearly 800 of these were allotted to me as Chef de Service," says Gama, "and I found them in a large house lying on what may more properly be called dung than straw, pressed one against another, uttering piercing shrieks, complaining with many tears of the way they had been forsaken, praying for death, for, when suffering from horrible tortures, existence is no longer a benefit. . . . No system, scarcely any nurses, no proper place where one could make broth for more than twenty-five persons, and there were no cooking utensils in which it could be boiled in the open air, no jugs from which to give drink to men agonised by insatiable thirst arising from the burning fever which their wounds provoked. In want of means and assistance, meeting no one with whom I could consult as to the best way to overcome the difficulty, obliged to prepare dressings with the bad and insufficient remnants taken from the field hospital equipment waggons, so little was the proper treatment of the soldiers of Napoleon then understood! Five thousand wounded men were lodged in several other houses which were called hospitals; they there received similar treatment, the negligence being only a question of degree. Yet Thorn is a commercial town advantageously placed on the Vistula, in the midst of a fertile country."

It may be interesting also to read the personal reminiscences of General Marbot, an officer of dis-

tinction in Napoleon's army, whose account, when he was wounded at the siege of Saragossa in 1808, gives us a picture of what he had to endure.

"At first I felt no pain," he writes, "and thought the adjutant standing near me had accidentally pushed me; but the blood soon came gushing out, and I found I had been struck by a bullet on my left side, and close to the heart! The adjutant helped me on to my feet and got me into the cellar in which the riflemen were, for I was losing so much blood that I was fainting. As there were no stretchers the soldiers placed a rifle under my arms and another under my knees and in this way they carried me through the ruins of this vast district to the spot where I had left General Razout. There my senses returned to me. The General wished to have my wound dressed, but I preferred to have it done by Dr. Assalagny, and after stopping my wound with my handkerchief I got them to carry me to the headquarters of Marshall Lannes, a cannon range from the town, in a huge abandoned inn. When the Field-marshal and my friends saw me arriving covered with blood and carried by soldiers, one of whom supported my head, they thought that I was dead. Dr. Assalagny assured them that it was not the case, and hurriedly attended to my wound; but they did not know where to put me as all the furniture belonging to the inn had been burnt during the siege, and there was not a single bed left. So they placed me on the bricks with which the rooms were paved. The Field-marshal and my comrades promptly piled up their coats and on these I was laid. The doctor examined my wound and discovered that I had been hit with a flat projectile, which had passed between two ribs without injuring them, which would not have been the case with an ordinary bullet. To find this projectile Assalagny probed the wound but found nothing. His face wore an anxious expression, and hearing me complain of extreme pain in my loins he turned me over on my face and started to examine my back. Hardly had he touched the point where the ribs join the spine than I was unable to restrain a cry of pain—the projectile was lodged there! Assalagny then armed himself with a bistoury, made a deep incision and saw a piece of metal lying between two ribs. He tried to extract it with his forceps, but, finding this impossible, in spite of such violent efforts that I was raised from the ground, he told one of my comrades to sit on my shoulders and another on my knees, and succeeded finally in extracting a large bullet, which the fanatical Spaniards had shaped like a coin, flattening it with a hammer. A cross had been engraved on each surface, and notches encircled it, giving it the appearance of the wheel of a watch.

"It was these notches which had hooked themselves into the muscles and had made the extraction so difficult. This flattened bullet was too wide for a rifle and must have been fired by a blunderbuss; striking me horizontally, it had cut like a sharp instrument and had passed between two ribs, twisted inside my body and come out in the same manner, luckily reserving enough velocity to pass through the muscles and the flesh of my back.

"The Field-marshal took the bullet to the Emperor to show him with what fanatical instruments the inhabitants of Saragossa defended themselves. After he had examined it, Napoleon sent it to my mother, telling her that I was to be made a major.

"Dr. Assalagny was one of the foremost surgeons of the time, and, thanks to him, my wound, which might have been fatal, proved to be one of the first to heal. The Field-marshal possessed a collapsible bed, which followed him in all his campaigns, and he was good enough to lend me a mattress and some sheets; my portmanteau served me as a pillow and my coat as a blanket, but in spite of all this I was most uncomfortable, for the room having no doors or windows, both the wind and the rain drove in. In addition to this, the ground floor of the inn was used as an ambulance station, and I had below me a great many wounded, whose groans increased my misery.

"The nauseating odour emanating from this temporary hospital reached to where I was. More than 200 canteen women had established their stalls round the headquarters; a camp was near by, and shouts, songs and the beating of drums never ceased. To complete this abominable chorus, the bass was played by the booming guns, whose fire never ceased day or night. I could get no sleep. I spent a fortnight in this plight, when my healthy constitution reasserted itself and I was able to get up."

The wounded had often to lie for days on the field of battle, where they had fallen, unattended, dying from exposure, pain, thirst and hunger;

thousands, whose lives ought to have been saved, perished in misery, abandoned by those who were responsible for their welfare. Surgeons and medical appliances were present, but in numbers ridiculously out of proportion to the needs of the situation.

Contemporary writers wrote of this intolerable condition of things as unavoidable, for no one, they maintained, could provide for so many evils to be remedied at the same time. It is a coward's attitude to regard such suffering as unavoidable. It is for those of us who have witnessed the miseries of warfare to guard against suffering, which can, and must, be avoided. But many years passed and many battles were fought before matters were improved.

It is no use lingering unduly on the waste of good material and the magnitude of human suffering for which an inadequate medical and nursing staff were responsible. But we come now to a chapter in history which brought matters to a climax, and although an equally unsatisfactory condition of affairs existed before the Crimean war, people took a livelier interest in this war, partly because it was nearer home, and greatly because in it we had our first great war correspondent, William Howard Russell, who opened our eyes to a state of things which the military authorities, curiously enough, regarded as inevitable.

Although England should have learnt, from experience in her wars in India and the Peninsula, the art of military administration in its many branches, it was found, on the outbreak of hostilities in the Crimea in 1853, that this department of State was in a deplorably inefficient condition, owing to the fact that it was under divided rule, the Minister of War being also, and principally, Minister of the Colonies, while the Secretary of War was almost entirely concerned with the duty of keeping military expenditure within limits sanctioned by Parliament. Besides these two branches there were many other military departments, the Horse Guards, the Ordnance Department, officials concerned with pension pay, transport, and many other kinds of business connected with war, who either co-operated or fought amongst each other, but seldom appealed to any paramount chief. This disastrous confusion had been brought about by the struggle between the monarchy, which tried to usurp personal power over the army, and our parliamentary system, which insisted upon the Ministers of the Crown being responsible for the conduct of affairs. As a consequence, every department proved itself to be wanting when the crisis of war arose, and no department suffered more, or broke down more completely, under the strain put upon it, than the Army Medical Service.

That admirable historian, Kinglake, in his 'Invasion of the Crimea,' says that the people of England had, in all matters dealing with war, fondly accustomed themselves to lean on the great shade of Wellington, but this idle habit was specially dangerous when applied to the care of the sick and wounded, for their treatment in the Peninsular campaign, where the death-rate was enormous, should have served rather as a warning than as a model.

The head-quarters staff of the Army medical department consisted, in 1853, of a director-general, one assistant, and about six clerks. Dr. Andrew Smith, Director-General at the outbreak of the war, was an able man, but he acted in subjection to at least five other departments, and his power to command hardly extended beyond the six clerks at their desks. State parsimony had brought the conditions of our Army medical service down so low that independence, courageous statement of opinion or enterprise were almost non-existent. The highest ideal of the Army surgeon seems to have been a stoical resignation to a condition of things which no man with any sense of responsibility or pride in his profession should have tolerated for five minutes.

The consequence of placing our sick and wounded in the care of such abject and hide-bound servants of a penurious and ignorant department was a waste of life and health among our troops, which, during the winter of 1854, almost threatened to exterminate our army in the field.

The task the Army medical department had to perform was to create suddenly, at the outbreak of war, every detail of a complicated system, and this had to be done with grossly insufficient funds, with practically no authority to command and with no trained staff.

There is no doubt that the Director-General was an able and experienced man, but he was trying to carry out his work under impossible conditions. Here is an instance: each time he wished to send to hospitals at the seat of war some neces-

sary, such as sago or wine, he must inform the Horse Guards, and the Horse Guards must move the Ordnance, and the Ordnance must instruct the Admiralty, and the Admiralty must give orders to the Victualling Office, which must make arrangements with the Transport Office, who must appeal for the assistance of private ship-owners to carry the article to its proper destination. But even in his attempts to prepare for the eventualities of war, the unfortunate Director-General was snubbed in the carrying out of almost every scheme which he was wise enough to propose. He submitted plans for the careful removal of the sick and wounded by sea transport, for ships to be in readiness for the reception of patients; he wished to establish hospitals in certain spots, he asked for competent men from the Army to act as hospital orderlies; he begged that pensioners should not be the only attendants upon the sick. It is almost incredible to read that every one of these very moderate demands was either entirely ignored or absolutely refused. The responsibility for such blunders was divided among so many chiefs and so many departments that it was impossible to fix the blame on any one offender.

The seat of war, meanwhile, was such as to make it quite certain that the capacity of the hospitals and their staff would be taxed to the utmost. Camped on the exposed Chersonese heights overlooking Sevastopol, they were sheltered from the rigours of a long Russian winter by only single canvas tents, with no bedding but the bare earth, sometimes frozen, often drenched. Their food was insufficient and lacking in many of the constituents we now know to be necessary for health, their clothing was grievously insufficient, and in the bitterest weather they never had more than one blanket apiece. A great many of these hardships were caused by a destructive hurricane which swept over the camp and the neighbouring harbour on November 14th, 1854, sinking ships laden with clothing, blankets and provisions, and the effects of this disaster were never quite obliterated.

Our French allies meanwhile rejoiced in a properly organised war department at home, which had provided an efficient ambulance corps for the sick, comprised of skilled directors, medical officers, and, above all, well-trained hospital orderlies and an equipment for conveying the wounded soldier from the battle-field to the well-ordered hospital in the rear. They started far ahead of us, but the insufficiency of medical officers and stores proved a fatal obstacle to the success of their arrangements. As the condition in the English camp improved, that in the French grew worse and worse. Thus, in a report from Inspector Baudens to Marshal Pélissier, dated March, 1856, he writes:

"It has not been possible to place our ambulances upon a good system. Blankets are numerous, but nearly all of them are contaminated, sheets are wanted, as well as the means of washing them. Many of the patients are obliged to sleep in their trousers, our field hospitals are in need of cots, utensils, jugs, spittoons, etc. . . . Still more so, slippers and hospital clothing." Again, Dr. Scrive, Médecin en chef of the French army, wrote: "Tents did not suffice to protect the unfortunate patients from the intense cold, and their toes were, in consequence, frost-bitten, almost without their being aware of it, and sometimes mortification of the whole foot ensued. It was a sad sight, but, in the absence of everything, what preventive remedy was it possible to find for these evils? We could only offer moral aid."

If the wretched men survived these first days of agony and were transferred to ships to carry them from the Crimea to the convalescent hospitals in Turkey, they found such ships absolutely unfitted for the purpose, and many poor fellows died from exposure and want of care. On disembarkation, "the fresh arrivals," writes Dr. Chenu, "wounded, suffering from diarrhœa, dysentery, and cholera, and who are brought to fill up the vacant beds, are landed at one of the points in the Bosphorus, and thence they are brought up on stretchers. They come from Kamiesh; they have had a voyage of three, four, or five days; they are in a pitiful state, covered with vermin, and suffering in every conceivable manner. Some are unable to speak, and to tell that their clothes contain their excreta since the day of their embarkation. The wounded are in a still worse state. They have not had their wounds dressed since their departure from the Crimea; the bandages are out of place, and do more harm than good; the swollen parts are being irritated by the hardened linen; mortification, and even vermin, have attacked the wounds; the odour which they diffuse is horrible, and would infect the wards if they were not kept in the open air, on the threshold of the hospital,

in order that the infected clothes might be removed, and the wounds washed and temporarily dressed before placing the poor wretches on the bed which the same morning was occupied by a comrade sent off to Gallipoli or France, or who, perhaps, died during the night." There was no lack of devotion on the part of the over-worked surgeons or orderlies, but human strength could not accomplish a task for which five times the number of willing hands would hardly have sufficed.

In the terrible winter which followed the outbreak of hostilities, the apathy of the British public was aroused by letters appearing in 'The Times,' written by Russell, which spoke in no measured language of the chaos existing in the British camp, and of the incompetence of those who were responsible for the welfare of the troops. As early as April, 1854, he noted the beginning of chaos in the commissariat and medical arrangements. "The management is infamous," he wrote, "and the contrast offered by our proceedings to the conduct of the French most painful. Could you believe it, the sick have not a bed to lie upon? They are landed and thrown into a rickety house, without a chair or table in it. The French, with their ambulances, excellent commissariat staff and boulangerie, etc., in every respect are immeasurably our superiors."

But meanwhile, how were the English placed? We have seen that for the care and treatment of ths sick and wounded they had in readiness no organisation whatever. The general in command of the troops was expected to arrange for all that was necessary, but to rely on such service from a man with the whole weight of the campaign on his shoulders was to court disaster. The arrangements made for our sick soldiers were absolutely insufficient and primitive.

Surgeon-Major Quesnoy, of the French army, reports that after the battle of the Alma the means of transport in the English Army was disastrously insufficient. "The wounded," he wrote, "passed two nights on the field of battle, and the greater part only received medical attention when they reached the ships which were to convey them to Constantinople." When the Army settled down for a winter campaign on the exposed plateau of the Crimea many necessaries for the care of the wounded ought to have been thought out: first, stretchers and bearers to convey him from the battlefield to the marquee which served as field hospital; from there he must be carried in some sort of conveyance to the coast, and thence embarked in a ship which should carry him across the sea, a journey of several days, to be disembarked, and eventually taken to one of the base hospitals established on the shores of the Bosphorus. So long and troublesome a journey would cause anxiety under any circumstances, but when it meant dealing with a constantly increasing stream of helpless sick the task became overwhelming, and the arrangements made proved wholly inadequate. By the end of October, Lord Raglan, Commander-in-chief, had spoken of his soldiers as "worn out by ceaseless toil, and infested with cholera" and other diseases, but battles, the effects of the bitter winter and cruelly excessive fatigue, had worked such havoc among them by the end of February that it was calculated by Kinglake that there were "lying in hospital beds, or else in new hospital graves, dug, all of them, within four months, a number of sick and dead together amounting to 22,506."

Russell puts the numbers of the sick much higher. In December it was reckoned as 19,479, and in January, 1855, as 23,076, and seven eighths of these cases were from sickness due to exposure and privation; one eighth only was due to wounds. To quote Russell's own words: "Rain kept pouring down, the wind howled over the staggering tents; the trenches were turned into dykes; in the tents the water was sometimes a foot deep. Our men had neither warm nor waterproof clothing; they were out for twelve hours at a time in the trenches; they were plunged into the inevitable miseries of a winter campaign." These were hard truths, which, sooner or later, came to the ears of the people of England. It was right they should know that the beggar who wandered the streets of London led the life of a prince compared to the British soldiers who were fighting for their country, and who, we were complacently assured by the home authorities, were the best appointed army in Europe. They were fed, indeed, but had no shelter. The tents, so long exposed to the blaze of a Bulgarian summer sun, and drenched by torrents of rain, let the wet through like sieves. Hundreds of men had to go into the trenches at night with no covering but their great coats, and no protection for their feet but their

regimental shoes. Many, when they took off their shoes, were unable to get their swollen feet into them again, and they might be seen bare-footed, hopping about the camp, with the thermometer at 20 degrees, and the snow half a foot deep upon the ground. The trenches were two or three feet deep with mud, snow, and half-frozen slush. It must not be inferred that the French were all healthy while we were all sickly. They had dysentery, fever, diarrhœa and scurvy, as well as pulmonary complaints, but not to the same extent as ourselves, nor to anything like it in proportion to their numbers. On January 8th some of the Guards of Her Majesty Queen Victoria's Household Brigade were walking about in the snow without soles to their shoes. The warm clothing was going up to the front in small detachments. We were astounded on reading our papers to find that on December 22nd London believed that coffee issued to the men was roasted before it was given out. Who could have hoaxed them so cruelly? Around every tent were to be seen green berries, which the men trampled into the mud and could not roast. I do not believe, at the time the statement was made, one ounce of roasted coffee had ever been issued from any commissariat at all to any soldier in the Crimea. There was a white frost on the night of January 22nd, and the next morning the thermometer was 42°. A large number of sick were sent on to Balaclava on the 23rd on French mule litters. They formed one of the most ghastly processions that ever poet imagined. Many were all but dead, with closed eyes, open mouths, and ghastly faces, they were borne along two and two, the thin stream of breath, visible in the frosty air, alone showing they were still alive. One figure was a horror—a corpse stone dead, strapped upright in its seat, its legs hanging stiffly down, the eyes staring wide open, the teeth set on the protruding tongue, the head and body nodding with frightful mockery of life at each stride of the mule over the broken road. The man had died on his way down. As the apparition passed, the only remark the soldiers made was, 'There's one poor fellow out of pain!' Another man I saw with the raw flesh and skin hanging from his fingers, the naked bone of which protruded into the cold air. That was a case of frost bite. Possibly the hand had been dressed, but the bandages might have slipped off. Yet people told us it was 'croaking' to state facts, or even to allude to them! The man who could have sat calmly down and written home that our troops were healthy, that there was only an average of mortality, that everyone was confident of success, that our works were advancing, and that we were nearer the capture of Sevastopol than we were on October 17th, that transport was abundant, and the labour of our army light, might be an agreeable correspondent, but assuredly he would not have enabled the public to form an accurate opinion of the real state of affairs in the camp before Sevastopol. The wretched boys sent out to us were not fit for powder. They died ere a shot was fired against them. Sometimes a good draft was received, but they could not endure the long vigil and exposure in the trenches."

There is no doubt that Russell's letters in the 'Times' profoundly affected the people who read them. He was the soldier's best friend, and the letters brought, presently, not only instant relief in the shape of comforts and necessaries for the army, but searching inquiries and reforms. "In my view," wrote Sir Evelyn Wood, writing to Russell years after," you did much more—you saved armies of the future by showing up our incompetence for war." Lord Aberdeen's ministry was turned out on Mr. Roebuck's motion for an inquiry into the state of the British army before Sevastopol.

When the worn-out sufferers, numbed by cold and half starved, succumbed to disease or were wounded, there awaited them the unspeakable sufferings of the rough field hospital, to be followed by transport for embarkation at the little port of Balaclava. Before this could be carried out there was often endless delay to be encountered, owing to lack of men for the duty. Even when this hardship had been overcome there followed a voyage of some 300 miles in ships absolutely unprovided with proper hospital arrangements, resulting in such horrible suffering for the patients that, in the months of December, 1854, and January, 1855, eighty-five, and, later, ninety out of every thousand among the sick and wounded died and were thrown overboard.

When the survivors landed there followed further painful delay on the beach before they were received into those hospitals which, instead of being harbours of peace, with a promise of renewed health, soon became notorious for the horrible conditions in which the care of the hapless soldier was carried on.

There can be no doubt that so much misery among the sick and wounded in war was no fresh evil. The Crimean war came after a prolonged period of peace, and whilst the work of civilisation had grown apace and men and women had come to have a greater regard for human suffering and a more intense desire to help all forms of it, the peace had led to a lamentable condition of drifting among the military authorities, a condition which is very apt to arise among us islanders, and to which we are in constant danger of succumbing.

The calamity of a great British war seems a very unlikely thing to most of us, and it is a little difficult to realise the necessity of being ready in every particular for the remote possibility of a big campaign. And yet the folly of a nation which refuses to be prepared for war can only be compared to the folly of the individual who refuses to make his will because death seems to him such an unlikely prospect. To make his will will not bring death one hour nearer; of the preparedness for war we can say even more, it will make its likelihood a great deal less.

It may be argued that preparation for war lies outside the sphere of most of us, but I want to prove to you that this is not so. The lesson we have learnt from former wars of which we have any details regarding the medical history, is this: that no government in the world can afford to maintain a medical or nursing staff or provide medical comforts sufficient to relieve the necessity of the sick and wounded in any war comprising even a few big battles.

When they are fought the medical service must appeal to the charitable and the competent to lessen the horrors which are inevitable if speedy and sufficient help cannot instantly be supplied.

In my next lecture I am afraid I must again harrow your feelings by completing the medical history of the Crimean war, but it will show you from what direction help came, and how it indicated to succeeding generations where they must look for help, and, above all, in what manner they must be prepared.

It is no new invention to try and mitigate the suffering of war by charitable assistance, but such assistance has failed, time after time, because it was unorganised, untrained, and, consequently, unskilled. To depend upon such help is folly; voluntary aid, organised in times of peace, is worth ten times the efforts put forth under the stress of emotion and in the rush and confusion of war.

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November 18th, 1912.

Pituitrin Therapy in Peritonitis.—Klotz refers to the vascular paralysis, especially in the splanchnic area, which is attendant upon peritonitis. Heart failure is secondary to this condition. The resulting fall of blood-pressure and paralytic ileus are of such character that pituitrin seems to be the logical indication. Still another reason for its exhibition is the ischuria. The author has tested the drug in twenty cases of peritonitis, all of which have been reported elsewhere. While some desperate cases seem to have been saved, the author's only inference is that the experience is sufficiently encouraging for an extensive trial. The cases most suitable are those of post-operative peritonitis where drainage is possible and other local measures can be carried out. As soon as the affection develops the abdomen should be reopened, the peritoneum irrigated and drained and pituitrin injected into the veins. Pituitrin acts opposite to hormonal, one of its competitors in paralytic ileus, in that it tends to raise instead of lower the blood-pressure. The latter attribute contra-indicates hormonal in peritonitis. — *Med. Record*, vol. lxxxii, No. 16.

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A LECTURE

ON

SOME POINTS IN ANO-RECTAL SURGERY.

Delivered at St. Mark's Hospital for Diseases of the Rectum.

By ASLETT BALDWIN, F.R.C.S.,

Surgeon to the Hospital; Surgeon to the West London Hospital.

GENTLEMEN,—It is pain which brings many people to this hospital, and many of the lesions we have to deal with here are no doubt extremely painful. The three which I should like to speak about first are anal abscess, anal fissure, and thrombosed piles.

To take the first, anal abscess, many of these cases come here with very great pain, and with the history of not having slept properly for several nights. We find that they have generally been treated by poulticing. Many also come when the abscess has burst into the bowel and a fistula is already present. But in a number that stage has not been reached. What is required in this stage is to operate on the abscess in such a way as to prevent the formation of a fistula. For that purpose I generally use local anæsthesia for men, gas more commonly for women. I get the women to lie on the side on which the abscess is, so that it is downwards, the men are usually in the knee-elbow position. The parts are disinfected with lysol lotion, of the strength of a drachm to the pint, and the skin injected with novocaine 2 per cent. with a few drops of adrenalin in it. To make the prick absolutely painless, if the patient be a nervous one, we spray the skin with ethyl chloride, and when the skin becomes white the needle is pushed just into the skin, but no further. Then the solution is very slowly injected, and as the parts become white the needle is gradually passed on. The amount to be injected depends

upon the size of the abscess. Some of these abscesses are quite large; I had a very large one to deal with last Tuesday. The abscess has to be opened in such a way that it will not readily close up. In some of these abscess cases there are large cavities, and if they are opened by slitting up the skin only in a direction radiating from the anus, the skin wound will close too soon, that is, before the abscess cavity has granulated up. You will notice that I speak of anal abscesses; I have not used the term "ischio-rectal," because they are different. The abscesses I speak of are at the anal margin and do not burrow into the ischio-rectal fossa. If you get the finger through the anus into the bowel you can feel a swelling in the ischio-rectal fossa in the case of ischio-rectal abscess; but that is not so in anal abscess. Most of the cases are anal, not ischio-rectal. Having rendered the part anæsthetic, the needle is inserted until the whole of the line is rendered white. Then the needle is again inserted at right angles to the first line of injection about its middle. In about a minute you will find that the parts are anæsthetic enough to operate. Then the point of a curved sharp bistoury is inserted at the outer margin of the abscess and is passed deeply and brought out just external to the anus and the knife made to cut outwards.

The old-fashioned radial incision is not enough for the purpose. The knife is therefore passed in at the centre of the incision, and cuts downwards in one direction and upwards in the other direction; that is to say a cut is made at right angles to the first, or, like it, the cut may be made by transfixion, thus making a crucial incision. But even that is not sufficient. Therefore, the points of skin formed by the crucial incision are cut away. To do that easily you grasp these points of skin with toothed forceps in your left hand, and with the right hand cut them away with scissors; by the time you have done a considerable area of skin has been removed. You have got it well open now, and it may take ten days to close, perhaps longer, perhaps less, according to the amount of skin removed. And, in turn, the amount of skin removed depends on the size of the cavity with which you have to deal. You can estimate the size of the cavity by gently passing a probe in different directions and feeling its extent. For a few days the part is treated with lysol fomentations and afterwards ammoniated mercury or eucalyptus ointment is applied. In many cases the abscess will heal up soundly and no fistula will be formed. In some cases where there is a great deal of spasm of the sphincter and of the levator ani, and the patient is in very great pain, I give him gas, and do exactly the same operation, at the same time dilating thoroughly the sphincter with the fingers. When the patient wakes up from that little operation he has no pain whatever. To enable you to do it under gas I should like to tell you of the method of giving that anæsthetic for anal operations. The patient is put on his side, the right hand or the left hand according to where the abscess is, the affected buttock being below, and the knees are thoroughly flexed. Round the knees and round the neck is put a jack towel, in order to prevent the patient straightening out during the procedure. Trussed up in this way by the jack towel, the patient is kept in the right position. Most jack towels are too long for this purpose, and allow some extension of the thighs. Then the towel is sewn across five or six inches or so from its end before use. Unless you use the towel you cannot very well carry out any operation under gas. A thrombosed pile has a different appearance although of course they may occur in exactly the same situation as anal abscesses. At first they are fluid swellings and soft to the touch and instead of being red are bluish in appearance; later on one can often feel a hard clot inside. The best treatment is to disinfect them and inject them with the novocaine and adrenalin solution, and slit them up by means of a radiating incision, not, in this case, a crucial one; and the clot is then exposed to view. It may be more or less adherent, or may turn out easily. It is not advisable to incise these unless you can feel the clot is present. In some cases the blood is evidently fluid when the patient comes, and if you incise when that is the case continuous pressure may be required to stop the bleeding. Having incised them you seize the clot with the same toothed forceps and draw it out. The clot is often due to rupture of a small vein in the pile, and the history often is that while the patient is straining, such as at stool, or lifting a weight, he has a sharp pricking sensation in the anus, and then the swelling appeared. But sometimes there is no clot in the tissues, but instead you find several tortuous veins, all thrombosed. Then

you can dissect all of them out through the same opening, while its edges are held apart with small hooks if necessary.

The after-treatment consists of putting on a pad of gauze, a piece of cotton-wool, and a T-bandage, telling the patient to, if possible, avoid opening the bowels for twelve hours. Before the bowels act I give the patient ammoniated mercury ointment to smear on the part, and after the motion the patient is instructed to wash the part with water, and dry with cotton-wool, and then put on some of the same ointment, and another small pad of cotton-wool, and outside, a T-bandage. With this treatment most thrombosed piles heal by first intention, and cause no further trouble. The disadvantage of leaving these piles unopened is that the clot is likely to become infected by the colon bacillus, if not other organisms, and a small abscess results, which, if left, may form a fistula; or if that does not take place the clot organises and may form a hard fibrous nodule, or may calcify and become a source of constant irritation. Sometimes it will cause pruritus. There are many people who are scarcely ever clean, and the pile will be a source of worry to them, but if it is dealt with in the way I have indicated, it shrivels up and no further trouble ensues. It is not uncommon for the patient to come back in a few weeks with one or more similar piles on the same or on both sides. They should be treated in the same way, and there is a probability that that will end their trouble. It is necessary to warn these people to avoid straining, and to see that the bowels are opened easily, because it is generally the straining in defæcation which is the cause of this condition. Many patients come here giving a history of constipation, and they say that when they do get the bowels to act they suffer from severe burning pain in the part, which lasts four or five hours afterwards, and sometimes all day. This generally means that the patient has got an anal fissure. There is usually an ulcer situated posteriorly just within the margin of the anus, and in many cases at its lower end there is a small rounded tag of skin called the sentinel pile. The amount of suffering that these little ulcers are capable of producing is out of all proportion to the size of the lesion. The reason of it is that one or more nerve-fibres are exposed in the floor of the ulcer, and these nerves are constantly irritated by the spasmodic contraction of the sphincter, and of course they are very severely irritated when defæcation takes place. In order to treat these it has been for many years the fashion with some to take the patient into a hospital or nursing home, give a general anæsthetic, and cut through the floor of the ulcer, dividing at least half the sphincter. Many times the whole sphincter has been divided. That, of course, cures the trouble. If there is a sentinel pile, it must be removed at the same time, and it is very important to investigate as to whether there is a polypus at the upper end of the fissure. If there is anything of the kind, that must be removed also. For some time past I have treated the great majority of these painful fissures without any operation and most of them heal. Before making an examination of these cases the history causes you to suspect the condition, and when you come to inspect the part you find the sphincter very tightly contracted, so that when you try to open the anus in order to look posteriorly you see absolutely nothing of the fissure. In those cases it is necessary to coax the patient to bear down slightly, but even then you may not be able to see anything. It is then advisable to blow some orthoform into the opened anus and wait for the effect. Anæsthesia is soon produced, and with the patient straining down a little, you can coax the part open with your fingers and then you can see the lower end of the fissure. A little more orthoform is blown in, and in that way you find the sphincter will relax and allow you to see the whole of the fissure. If the fissure is not associated with a sentinel pile, the treatment I adopt is to apply thigenol on a piece of cotton-wool to the fissure. Thigenol is a synthetic product similar to ichthyol and is about half the price. It does not stain the linen, and has no objectionable smell, and does not produce pain when applied to ulcers. You will realise that those are very important points, and they make thigenol very valuable for these conditions. To apply it I wrap a little wool round a probe, and dip it in the thigenol and carefully insert the wool, which remains wrapped round the probe, within the anus, and press it gently on to the ulcer; then with the index finger of the left hand I hold the wool, and gradually withdraw the probe, leaving the wool saturated with thigenol in position. That is only part of the treatment, for you will have to see that the patient's bowels are open every day without

straining, and that the motions are soft. To do that, I get the patient to drink every night a tumblerful of water, either hot or cold as he wishes, and an hour before breakfast in the morning another tumblerful of water, hot. And to ensure the softness of the motions and their easy passage, I give petroleum four times a day. That is used in large quantities now in treating rectal lesions. The particular formula which we use here is tincture of cochineal 10 ♏, essence of peppermint 10 ♏, syrup or glycerine 1 drm., paraffin mollis 1 oz. It is better to speak of paraffin mollis rather than vaseline, because the very idea of vaseline would put some people off the mixture, and they would not take it. If they find it objectionable it is well to add 20 gr. of powdered cocoa. That makes quite a pleasant preparation of it, and the cochineal and peppermint may be omitted. If there is a complaint that it is too smooth to the mouth you can add 10 to 20 gr. of powdered agar, but the majority of patients take the mixture very well. Where there is objection you may order, instead of the paraffin mollis, parolein, but they generally require a little more of that during the day. The first prescription I gave you should be taken four times a day, a teaspoonful each time. Numbers of London chemists make excellent preparations of this kind. A very good one is made by Cooper, Son & Co., and called "aperol"; it is about of the consistency of vaseline. They have another preparation called "aperol liquid," of the consistency of parolein; they also put the latter up in capsules. People who cannot take the above will take the capsule form quite well. It is not expensive, and the flavouring makes it agreeable. These patients having been very constipated before you see them, the petroleum is not sufficient to get the bowels to act; it is therefore necessary to give them some aperient in addition. Confection of senna is frequently prescribed here—half a teaspoonful, or up to one teaspoonful, according to the need of the case. After the bowels have acted, the patient is instructed to wash the parts with water, to dry afterwards with cotton-wool, and then apply some unirritating ointment such as eucalyptus. Many of these cases will heal with this treatment. It is a great score to be able to get them healed without an operation, because you know many people entertain the greatest objection to having their sphincters partially or wholly divided. I do not say all cases should be treated in this way, but there are some people who come with violent spasm of the sphincter, with a sentinel pile, and in very great pain indeed. To such patients I give gas, place them in the position I have mentioned, truss them up with the towel in the way I have described, and then thoroughly dilate the sphincter with the fingers for about a minute. You will see the importance of having the patient firmly trussed up in order to do this. When you have done that, remove the sentinel pile thoroughly with forceps, and see if there is a polypus. Mostly there is not. If there is, get hold of it with forceps and ligature it rapidly. Afterwards wipe the parts with lysol lotion, and then put on a pad and T-bandage. Most of these people come afterwards and say they have had no pain whatever. Some say they have had a little pain when the bowels acted for the first time after the operation. There are some cases in which the fissure is old and deep and the sphincter is exposed, and they may not be cured by this treatment. Such cases you must take in and divide the sphincter half way, and dilate the anus. Most of them, however, are cured by the simple means I have described. There is another ulcer which causes very great discomfort, though not so much as the one I have described under the name of fissure. This is an ulcer which occurs a little higher up, but in much the same situation. The pain in these irritable ulcers comes on generally an hour or so after the bowels have acted, but it does not last so long. Perhaps the pain radiates over the sacrum, as it will in fissure, and possibly also down the thighs and into the abdomen. Many of these also can be made to heal by giving them the same petroleum mixture, and by giving them water and confection of senna to make the motions soft, and by applying thigenol or some other non-irritating antiseptic locally. It is well to wash out the bowel once or twice a day with some substance such as chinosol 1 in 4000. These ulcers will not all heal under the above treatment, and so it will be necessary to take those that will not into hospital, give an anæsthetic, dilate the anus, and look round to see if there is any other lesion. If there is not, apply pure carbolic acid to the ulcer, see that the motions are kept soft, wash out with chinosol, and keep the patient lying down. Lesions about the anus heal up much quicker if the patient is lying down than if he is sitting or standing up. That is for the same reason that

ulcers of the leg heal up much quicker with the patient lying down, namely that the veins from the anal region have no valves, consequently the veins have to bear, when the patients stand, the weight of the whole column of blood from the heart. If the ulcer prove refractory, it should be ionised with 2 per cent. zinc sulphate solution, applied by the positive pole of a battery.

There is another important point which I should like to mention, and that is in connection with cases of chronic diarrhœa, or where there is the passage of mucus and perhaps of blood. That is to always have the rectum examined with the sigmoidoscope if there is nothing to be felt by the finger. Some people think that the sigmoidoscope is very dangerous to use, but it is very constantly used in this hospital, and I have never heard of any harm being done by it. If used with proper precautions an accident cannot very well happen. The object of examining the bowel is to see if there is any ulceration or malignant disease, because so frequently one or other, or both, of these lesions are present, and the trouble is thought to be only colitis. To examine the bowel properly with the sigmoidoscope the bowel must be empty. To bring that about you should give an aperient, such as castor oil, thirty-six hours previously. If the bowels are not acting well this should be given forty-eight hours previously, and another dose after twelve hours. The bowel should be washed out at least four hours prior to your inspection. If the washing is done nearer than four hours to the time of examination there may be some retained fluid, which will interfere with your examination. To examine with the sigmoidoscope you should first insert the index finger of the left hand into the anus, having well lubricated it with vaseline, and then put the point of the obturator of the sigmoidoscope on your finger, and as you withdraw your finger you push the sigmoidoscope with its obturator through the anus, so that as the finger slips out the sigmoidoscope slips in. In that way you can get the instrument in without producing discomfort, and you can pass it several inches without withdrawing the obturator, but when slight resistance is encountered the obturator is withdrawn and a small electric lamp is introduced, by means of which the interior of the sigmoidoscope and bowel are illuminated; then with the right hand you pump air into the instrument. The air distends the bowel in front, and by moving the sigmoidoscope in different directions you can see where the lumen is. When the lumen comes into view you push the sigmoidoscope onwards, but not until you see the lumen of the bowel do you ever press the sigmoidoscope forward. You therefore pass the whole of the instrument under the guidance of sight except at the very first introduction. You will soon discover if there is proctitis or abnormal redness of the mucous membrane, or ulcers, polypi, or stricture, or if the bowel appears to be fixed by adhesions. You find as you get higher up whether the bowel moves as easily as it should, because the bowel must be considerably movable before you get the instrument in. If there is a malignant growth, as you get nearer to it you may find more mucus, and probably blood. By the fact that the condition is seen to be worse as you get higher, you can often diagnose whether there is any gross lesion further on with comparative ease. Suppose you have excluded malignant disease, and have decided that the disease is ulcerative colitis, because you may have seen the ulcers, and the patient's bowels are perhaps moved ten or fifteen times a day, there is a high temperature, with rapid emaciation and he is extremely ill, medical treatment will be tried first. If one washes out the bowel with weak antiseptics such as chinosol 1 in 5000, or protagol 5 or 6 gr. to the pint, and that does not soon cause improvement, you should do appendicostomy at once without further delay, or the chances of recovery will be very small. It is usually quite easy to do. You cut down on the region of the appendix, two inches internal to the right anterior superior iliac spine, split the muscles in the direction of their fibres, introduce the right index finger, draw up the cæcum, find the appendix, attach the cæcum at the base of the appendix to the parietal peritoneum by means of a suture passed through the longitudinal band, and bring the appendix through to the surface, suturing the muscles and skin. The tip of the appendix is cut off, and a catheter introduced through it into the bowel, through which the irrigation is carried out. It is important to remember that these patients are becoming rapidly poisoned, and you must not waste time, or they will probably die.

In the remarks I have made I have assumed that the patients are very ill, and the choice of an anæsthetic is an important one. I think it is best

to give ¼ gr. of morphia and $\frac{1}{100}$ gr. of atropine one hour before operation, then you can do well with gas and oxygen. I do many formidable operations entirely under that anæsthetic. It is also important to remember when treating cases of colitis the value of vaccines. There was an excellent case of that kind brought under my notice some time ago by my friend Dr. Sydney Lee, of Teddington. He was fetched over to Switzerland to see a patient there, who had been treated by several local medical men for colitis, and had been given up, it being said that she could not possibly recover. Dr. Lee saw the girl and advised that a vaccine should be made and tried. The doctors there said it was not of the slightest use, and Dr. Lee had to overcome tremendous opposition. He sent some of the fæcal material to London, a vaccine was made as soon as possible and it was given to the girl. From that time she started to recover, and slowly proceeded to do so, and to everybody's astonishment, in a comparatively short time she was beginning to get about. Apparently she got well, but subsequently when in some more distant part of Europe she had a relapse and died. I have no doubt that had she been within the reach of Dr. Lee and under his care in that relapse she would have been alive to-day. I mention that to impress upon you the value vaccines may have in cases of colitis, and I hope you will remember also the importance of appendicostomy. Another point of importance which I should like to mention is brought out by another case, but of a different nature. About two months ago I was asked to see a young lady, æt. 23 years, who, several weeks previously, had had a baby, and in delivery the vagina and rectum had been torn into one. The rent was sutured, but broke out again, and when I saw her the fæces were all being passed through the vagina. She had severe pain on the right side of the abdomen, a very dirty tongue, and high temperature, and it looked very much like a case of appendicitis, and it was under that idea that I was sent for, with a request to operate if necessary. There was no definite localised lump over the appendix, but the right side of the abdomen was very tender and rigid and the tenderness and the pain extended to her right kidney. At the back over the kidney she was distinctly tender. But that did not exclude appendicitis, because I have frequently noticed it in appendicitis, apart from an abscess tracking up there. I have not seen that mentioned in books, but I have frequently noticed it clinically. I began to suspect, from the general appearance of the girl, the character and situation of the pain and the tenderness, that it might be due to a colon bacillus infection of the urinary tract. The meatus was swabbed carefully, and the water drawn off with a catheter. It had that peculiar dusky appearance which is found in *Bacillus coli* infection. We tested it with litmus paper, and found it acid, which tended to confirm our suspicion.

A microscopical examination proved the urine to be swarming with bacilli. She was too bad to justify our waiting to have her own particular bacillus grown for the purpose of making a vaccine, so we obtained Burroughs Wellcome's colon bacillus vaccine, and injected, as a first dose, 25 millions. From the first dose she started improving, and with the successsive doses that improvement progressed, and now she is up and about, all the symptoms having passed off; at the same time she had 30 or 40 grain doses of potassium citrate. In a short time I shall try to close the recto-vaginal fistula. That case simulated appendicitis very closely, and it would have been very easy to have been misled into opening her abdomen and removing her appendix, which would not have done her any good. It is important to remember that if women get symptoms simulating appendicitis during pregnancy, especially if the trouble is on the right side, that it may be due to *Bacillus coli* infection of the kidney, ureter and bladder. I could tell you of other very instructive cases of this kind, but they perhaps scarcely come within the scope of this lecture.

There is another point I want to bring to your notice, and that is the method of rapidly and easily doing colotomy and the method of closing it again. There are many cases in which a colotomy has to be done and where it must be permanent; but there are also a number of cases in which it is possible that it may be only a temporary measure. An incision is made through the skin at the junction of the outer and middle thirds of a line drawn from the umbilicus to the left anterior superior spine down to the external oblique aponeurosis. This is split in the direction of its fibres parallel to Poupart's ligament; the split edges are separated from the muscle below and drawn apart;

the internal oblique and transversalis are then split in the direction of their fibres. The internal oblique fibres run at right angles to those of the external oblique. In many cases when you have to do colotomy intestinal obstruction is present, and the bowel is partially paralysed owing to over-distension. It is important, therefore, in doing colotomy, that the loop of bowel which you have drawn out shall not be compressed, but there should be every inducement offered for an easy escape of the retained fæces. This is secured as follows: Having drawn the loop of the sigmoid out, select the highest part which you can conveniently draw down and return the rest into the abdomen. If you take the lowest part there will be considerable prolapse of the mucous membrane afterwards, causing much worry and inconvenience. Then you take a long curved needle threaded with silkworm gut, pass it through the middle of the outer side of the incision, then through the mesentery of the intestine, and half an inch lower down back through the mesentery again, and out again through the same side of the wound as at first and tie it; so there is a loop of silkworm gut passed through the mysentery, and through merely one side of the wound; this prevents compression of the bowel, and the mesentery is fixed to the wound, forming the spur which is necessary to prevent the fæcal material getting to the lower part of the bowel. This is the first and most important stitch of all. The second stitch is one taken through the upper angle of the wound and through the anterior longitudinal band of the bowel and tied. The third is passed through the lower angle of the wound and anterior longitudinal band in the same way. If you are going to open the bowel at once, you take a somewhat small curved needle armed with thread, and suture the bowel to the skin all round so that the peritoneal cavity is completely shut off. The suture should pass through the skin and as far as the submucous coat of the bowel. Before opening the latter I wipe round the junction with tincture of iodine, and tuck round some gauze as an extra precaution. To open the bowel I put in a purse-string suture parallel to the long axis of the bowel through the anterior longitudinal band, and make an incision through in the area surrounded by the suture, taking care not to cut the stitch, and pass in a Paul's tube, and secure it in the opening by tying the suture. On the end of Paul's tube is slipped a soft rubber tube, so that the fæcal contents are conveyed into a suitable vessel. This is a very clean and convenient way of doing colotomy, and does not dam back the fæcal material. If the bowel is to be opened in four days' time, a fourth stitch should be passed opposite to the first one mentioned, through the bowel and whole thickness of the abdominal wall to prevent possible prolapse of small intestine through the wound beside the loop of large bowel. With regard to closing a colotomy opening that is often a very formidable and dangerous operation, but if the colotomy has been done in the way I have described it is quite simple and safe.

To close it take a sharp knife, and make an incision all round through the skin, about $\frac{1}{8}$ in. away from the mucous margin. Now you have got the bowel separated from the skin, with a thin collar of skin attached all round. Now remove just its epithelial surface with scissors curved on the flat. Then take a curved needle armed with thin thread and pass it through this collar of skin from side to side and completely close the opening, the suture being a continuous one. To still further make this safe, put another continuous suture through the peritoneum, muscular coat and submucous coat of the bowel, and bury your first suture. With the handle of your knife carefully separate the bowel from the tissues on each side until you get well down, then with your knife or scissors cut absolutely closely to the skin for a very short distance at the upper and lower extremities of the loop to separate the bowel from the skin in these situations, where it is more adherent than at the sides. If by chance you open the bowel, sew it up again by a double layer of sutures, and then there is no harm done. You have now got the bowel separated from the skin all round. With the finger feel for the aponeurosis of the external oblique, and with your finger separate the bowel from the aponeurosis. The bowel is pushed below the latter, which is overlaped and sutured, and so the colotomy is closed. Suppose it is a person who will want to do muscular exertion afterwards, such as a man whose colotomy I closed some time ago, and who wanted to play football, you must then identify the internal oblique and transversalis muscles, sew them together with iodine formalin catgut, and then complete the operation as before. It is advisable to insert a drain-tube down the centre of the wound to near where you have closed the bowel; it need only go near the suture line. I have not yet had one give way, but it is well to leave the tube in until the bowels have been well opened, and if nothing untoward happens, take the tube out. You will see by this method the peritoneum is not opened at all, and if leakage should occur, it takes place outside the peritoneum and traverses the tube track, and need not cause anxiety, as it will gradually cease spontaneously.

November 25th, 1912.

THREE LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
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LECTURE II.

MODERN HISTORY: A.D. 1855–1871.

AT the outbreak of the war in the Crimea the British Government had provided no hospitals for the reception of the sick, no efficient ambulance corps, no vessel fitted for the transport of patients. A few medical officers were on the spot, but for the nursing of the sick and wounded there were only sergeants and soldiers lent from the ranks, with no training whatsoever, and, with the perversity which seemed at that time to characterise the authorities, these temporary orderlies were invariably recalled to their regiments as soon as they had mastered the elements of nursing, and were replaced by novices. Lord Raglan, when he took command of the Army, at once proceeded to establish general hospitals, and in time these were planted in various parts of the Levant, in the Dardanelles, in Rhodes and Smyrna, with some floating ambulances. But by far the greater number of patients were received in the hospitals at Scutari, and these overcrowded, insanitary and under-staffed institutions soon became homes of anguish, horror and death! Mattresses touched each other, and two persons could hardly pass in the same passage; it was considered dangerous to open the windows; everything was dirty; vermin swarmed; large buckets supplied the place of chamber utensils, and the approaches to the buildings were poisonous with filth.

From the first, it may be remarked, the two hospitals under the care of our naval authorities were well conducted, and were free from the worst evils which were such a cruel reproach to the military establishments.

Lord Raglan, anxious for the welfare of the hospitals, despatched his principal medical officer in October, 1854, to inquire into their condition, but I have said that our system of training the Army medical officers had resulted in their being worthy, stereotyped machines, subdued and drilled into a belief that the existing state of muddle and inefficiency was unavoidable, and he consequently reported that the state of the hospitals was "as good as could be expected." "Ill-salaried, ill-treated by the State," writes Kinglake, "schooled in the habits of resignation, and bending under a load of professional work, which they performed with a generous zeal, the medical officers acted as though there should be no discontent—as though, not only on behalf of themselves, but also on behalf of their patients, they ought to accept all the miseries which crowded in on the hospitals as dispensations resulting from war—dispensations to be borne with that silent, that soldierly fortitude which disdains the resource of complaint, and they even, indeed, went the length of refusing to acknowledge a want." This stupid submission to things as they were, this inability to struggle and clamour for better things, was a source of a great part of the evil.

The condition of the hospitals was shamefully insanitary, but it was not till the following spring, when a new power had arisen and better days were in store, that any engineering work was attempted to put these huge crowded buildings into any kind of sanitary condition.

Slowly the anger of England was kindled against the ignorance, apathy and official incompetence which sacrificed the lives of those who were fighting for their country. The patience under suffering displayed by our soldiers was above all praise, but as accounts of what they had to endure became known through the press and from many private sources, there arose in the hearts of some noble women a desire to act and to help, which could not be denied.

It must be remembered that, at the date of which I speak, women were less well-educated and in a position of greater subjection than they are now. They were regarded with very real veneration and with a sense of true chivalry by all right-minded men, but also as creatures from whom the rough side of life must be hidden, who must not be contaminated with what was gross and ugly. They were regarded, not so much as the com-

* Gresham Lectures delivered in October, 1912.

panions and fellow-workers of men, but as their better angels. It needed more than ordinary courage for ladies of gentle birth to lay aside their reserve, to come out of the almost conventual atmosphere in which they were treasured into the vulgar light of day as champions of reform. But to the courage which animated certain women we owe the fact that the treatment of our sick and wounded was revolutionised, and that never again was such an iniquitous system of mismanagement allowed to prevail in our military and civil hospitals. To the sagacity of Mr. Sidney Herbert (Lord Herbert of Lea) we owe the initial step of introducing women as nurses into military hospitals.

Very fortunately for England he was, at this time, Secretary-at-War, and he was gifted with that fine independence of thought, that want of official pedantry, which appeared to hamper and clog so many of the Ministers of the day. He possessed also a personality which made him beloved and trusted by others, and he was able to usurp an authority in all that had to do with the care of the sick and wounded which made him invaluable at this crisis.

He realised that the paramount need for the improvement of the hospitals was not so much medical stores, which a generous public was sending out, literally, by the ton weight, but nurses to tend the sufferers and to dispense those stores. He wrote to Miss Florence Nightingale, who at that time was presiding over the Home for Sick Governesses in Harley Street, asking if she would go to the seat of war in charge of a company of duly qualified nurses? His letter crossed one from her offering her services. Within a week she had selected a staff of thirty-eight women and had started for the East.

Some months later she was followed by forty-six voluntary and salaried nurses under the guidance of Mary Stanley, daughter of the Bishop of Norwich and sister to Dean Stanley. All these devoted women went to nurse, and they put their services under the direction of the medical authorities. Their influence was felt at once, and to whichever hospital some of these gracious women came, greater comfort, order and better management succeeded the chaos which had prevailed.

But something more than the gift of nursing (invaluable as such service then was) was needed at this juncture—a commanding brain, the genius of organisation, the contempt for petty authority, the sense of proportion which even good women so often lack, all these were imperatively necessary to sweep out the Augean stables of old-established evils, to let in the waters of a more liberal policy and of modern methods. And at her hour of greatest need England found one such person—a woman endowed with the gift of organising and governing, fearless, tactful, clever, and trained in the art she so helped to ennoble. I speak, of course, of Florence Nightingale.

To those in England who were chafing under the belief that military punctilio and foolish bureaucracy were chiefly at fault in blocking the way to reform, it seemed certain that a woman's influence would mean the breaking down of stilted rules and regulations, with a softening of military rigour, when dealing with such a mass of suffering. But to Florence Nightingale, trained in a school of hard work and obedience, it was obvious that discipline, unswerving and almost rigid, was essential in the management of immense hospitals and terribly large numbers. She is described as "of slender, delicate form, engaging, highly bred, and in council a wrapt, careful listener, so long as others were speaking, and strongly, though gently, persuasive whenever speaking herself; the lady-in-chief gave her heart to this enterprise in a spirit of absolute devotion."

Although often hampered by official inertness, she was loyally supported by Mr. Sidney Herbert at home, but it must be remembered that it took two weeks for a letter to reach London and two more before the reply could be received, and the electric telegraph, then in its infancy, only covered part of the distance, and was seldom employed. Thus she had to act independently, and her influence became paramount in her dealings with hospital management. Her soundness of judgment was admirable, and her active brain was able to seize facts and to form instant decisions in a manner which seemed marvellous to the slower minds of the men with whom she had to deal. She obtained a dominion over them which gave her the power she needed to institute reforms, generously admitted by them to be due to her "commanding genius."

The reforms she promoted were many, and when we find that among these was the very elementary need of establishing hospital laundries, one wonders

how such institutions were run at all before her administration began. In the big general hospital at Scutari, where she principally worked, "the authorities" had only succeeded in washing seven shirts during the previous month! Whilst her dominion over the minds of the officials was great, and she was loyally assisted and served by the medical officers, of whose zeal and devotion to their work she spoke in the highest terms, her influence on the unfortunate men stricken around her, and on overworked orderlies who had to serve under her direction, became proverbial. They toiled unweariedly and ungrumblingly at her request, yet "never," as she gratefully wrote, "came from any of them one word or one look which a gentleman could not have used; and while paying this humble tribute to humble courtesy the tears come into my eyes as I think how, amidst scenes of loathsome disease and death, there rose above it all the innate dignity, gentleness, and chivalry of the men (for never, surely, was chivalry so strikingly exemplified), shining in the midst of what must be considered as the lowest sink of human misery, and preventing instinctively the use of one expression which could distress a gentlewoman."

To the poor stricken soldier she became the guiding light, to which he turned for strength in his agony and for comfort when he was dying. Tender and womanly as she was to him, she soon became a power not to be gainsaid when she insisted on maintaining her authority over those working under her, and when determined to obtain what she knew to be right for her patients. Foul surroundings, filthy bedding, improperly cooked or bad food were no longer tolerated, and when official obstructors blocked the way she used her private resources to furnish new kitchens and other reforms which she knew to be essential for her patients' welfare. She came, a gifted and well-trained woman, with no official red-tape to bind her, and no ancient formulas to hamper her, and she swept the old abuses aside in a way which seemed miraculous to the hide-bound officials, who looked on in wonder.

She had to fight her way inch by inch through a dense forest of prejudice, but she fought with a woman's tact and patience and sympathy, and in time she broke down the ill-will of many who at first thought that a lady should not mix herself into matters so ugly, miserable and sordid as much of her work necessarily had to be.

She was ably supported by contributions which poured in from England, and she supplied the hospitals with some absolutely necessary furniture, with means to maintain cleanliness and with shirts for the patients. From the moment she interposed in the management of the hospitals order began to spring out of confusion, purity where there had been dirt, convalescence where there had been little else but death.

As a consequence of the reforms introduced and to measures taken at last to put the British hospitals on the Bosphorus into some kind of sanitary condition, the death-rate by the middle of March began to fall. Ventilation, cleanliness and a pure water supply did the work which a little forethought and energy should have done originally to prevent the horrible sacrifice of lives which had gone on through the winter. According to Kinglake the appalling total of 42 per cent. of deaths had been reached in February, in March it fell to 31 per cent., then 14, in April to 10 and then to 5, and by the end of June, 1855, it was only just over 2 per cent. —a rate of mortality exceptionally low for military hospitals in time of war.

The accounts which reached the British public by means of the press, and, primarily, from the pen of Russell, were a revelation to most men, and yet the evil was not a new one, for though we are apt to think that the Crimean war was peculiar in the cruel suffering it imposed upon its victims, there is no doubt that in previous wars the sick and wounded had suffered quite as much. And they might long have continued to do so if it had not been for the protest raised by the press and for the mission of Florence Nightingale.

Russell proclaimed the evil, Miss Nightingale proceeded to mitigate it. The English press was the only one to take up the cause, and the English Army at once profited by the knowledge it diffused, but it is gratifying to note that it was not the English Army alone which benefited by the experiences so dearly purchased.

Before the end of the war the influence of Miss Nightingale had completely changed the condition of the hospitals, and although, at the beginning of hostilities, the French system had been held up as the model to which we ought to aspire, yet two years later we can, without boasting, say that the cleanliness, sanitary condition and comfort of our troops were considerably better than theirs. Typhus, scurvy

and cholera still raged in the French and Sardinian camps, while these heralds of bad sanitation, bad food and bad water had completely disappeared from the British troops.

The danger still existed lest matters might be allowed to relapse into their previous condition of inefficiency directly the public mind was diverted and the war forgotten, but thanks to the energy of Mr. Sidney Herbert and to the enthusiasm of Florence Nightingale this was not so. This great woman, although broken in health from the hardships she had endured, suffered no relaxation in her determination to have the military hospital organisation put on a better basis. She became the authority on nursing and sanitary schemes to whom not only our own Government and military authorities applied for advice, but to whom foreign ministers and generals appealed for guidance on the outbreak of wars.

Our War Office requested her to draw up an exhaustive and confidential report on the working of the Army Medical Department in the Crimean war, and thanks very greatly to her energetic and tactful advice and to the interest she had awakened in the subject, this report led to the complete reorganisation of that service. Her influence led to the more sympathetic, more capable tending of the wounded in the two great wars which followed, the American Civil War of 1861–5 and the Franco-Prussian War of 1870–1.

It was the general enthusiasm she aroused in the lot of the sick and wounded which was the primary stimulus which led others to found the Red Cross Society, that international charity which concerns itself with the humane treatment of those who must suffer, directly and indirectly, so long as nations cannot live at peace, but must settle their disputes by the bitter test of war.

The founders of this society realised that the perfecting of the Army medical service is not enough. We have seen that the French sanitary service was well organised, and yet it came to grief. Ours was absolutely unorganised, and yet it triumphed in the end. Why? Because, not for the first time in history, the French authorities were taken by surprise at the magnitude of the disasters that came upon them, and their provision was wholly inadequate for the demand made upon it. England, on the other hand, after a disastrous beginning, found voluntary help to supplement that which the Government could provide.

Dr. Evans, an American by birth, naturalised in France, who, owing to the interest he took in the cause of the sick and wounded, visited the battlefields of Italy, America and elsewhere, affirms that "the accounts of *all* great battles prove that, during strategical evolutions, and especially after an engagement which has cost many lives, more men and a greater quantity of material are required for the immediate assistance of the wounded than any government has yet been able to place at the disposal of its army hospital corps." This must be admitted as an undisputed and incontestable fact, proved by every great war from the earliest days down to modern times.

The improvement in the Army medical service has been great, and continues to bring honour upon that fine corps, but in times of a national crisis we must look for something more, and it became evident to the philanthropic thinkers of the middle of the last century that voluntary assistance must step in and fill the gap.

Florence Nightingale's advice was asked concerning the Army medical service of India, and when the Mutiny broke out she nobly volunteered once more to go out and serve her stricken countrymen, but though the condition of her health would not allow the acceptance of this offer, there is no doubt that her example and counsel led to a more intelligent treatment of the sick in all our subsequent wars.

In spite of the suffering endured by the French soldiers during the Crimea, it was unfortunate that they profited very little by that lesson, possibly because they continued to rely too implicitly upon their official organisation, which was excellent on paper, but which failed so lamentably because it was inadequate for the demands made upon it by a great war.

Once again the French were to suffer cruelly from this same cause, although the expedition of France in 1859 to assist the Italians in their war of independence against Austria led to a great display of patriotic assistance which the Italians gratefully bestowed upon their allies. The enthusiasm of the inhabitants was shown by many improvised hospitals being established wherever the wounded were numerous, and comforts and attention were lavishly given both to friends and

foes. But the war proved exceptionally sanguinary, and the enthusiasm of a large number of charitably inclined bodies was checked by sheer inability to keep pace with the disasters. This want of sustained effort was due entirely to a lack of organisation and training. "The first excitement," as Dr. Evans wrote, "died away beneath the weight of difficulties which the general inexperience had accumulated." Wherever organisation did exist, as in the case of those Sisters of Charity who were present, the value of their services was past all words. But in the face of such numbers as fell, their services and those of the few whose sustained efforts tried to assuage the mass of suffering were but a drop in an ocean of misery.

Now there happened to be travelling in Italy, in the summer of 1859, a Swiss gentleman, Monsieur Henry Dunant, and he chanced to find himself in the immediate neighbourhood of Solferino on June 24th, a sultry day, followed by thunder and hail-storms, when the Austrians were beaten by the allied armies in a bloody battle. He was so deeply impressed with the events of that day and those that followed, that he published his impressions in a little book, 'Un Souvenir de Solferino,' which has since stirred the hearts of hundreds of readers of all nations, and has led to that improvement in the care of the sick and wounded for which he craved.

The armies, he tells us, encountered each other suddenly in the early hours of the morning after fatiguing marches on the previous days, and before any food had been taken or elaborate plans had been made for the formation of battle. They fell upon each other with a contempt for death which proved their heroism but led to the slaughter of thousands. Of killed and wounded in the Austrian and Franco-Italian armies there fell that day nearly 52,000 officers and men.

Admirable preparations had been made by the army medical service of France, but in the face of such numbers their efforts were horribly and ludicrously inadequate. Whatever casualties the army medical department estimates for, it is only for the men on their own side; the casualties of the enemy are seldom considered. Every large house, church and building in the neighbourhood becomes a temporary hospital, filled to overflowing with unspeakable suffering, for where could bedding, dressings, surgeons, nurses—everything that was necessary to save the lives of these thousands of fallen heroes—be procured at a moment's notice? There was not even water procurable to assuage the burning thirst of those lying helpless for endless hours under a torrid sun.

There was a sufficiency of food and charpie to dress the wounds, but a great lack of willing hands, for the few doctors on the spot were working day and night at countless surgical operations. A plague of flies, attracted by the decomposing wounds, bred maggots under the improvised dressings.

Thirty-six hours after the battle Dunant organised a voluntary band of women, aided by boys, as water-carriers. He concentrated his efforts on a church crammed with 500 wounded, while 100 others lay on straw outside the building. Water, soup and lemonade were in turn provided, while the neighbouring town was ransacked for linen, bandages, pins, cigars, tobacco and fruit. The tobacco was freely smoked, and served the double purpose of neutralising the odour of putrefying wounds and of solacing those who were undergoing operations. The wounded for the most part suffered or died like heroes. Many of them were attacked by pyæmia and hospital gangrene, which necessitated the secondary amputation of limbs, too often without chloroform.

The noble women of Castiglione treated the prisoners and wounded of every nationality alike, and called them all brothers.

Dunant saw wounded men bleeding to death hours after the battle had ceased, others being trampled under the wheels of artillery because there was no one to drag them into safety; hundreds more died from exhaustion two and even three days after the battle, because no one had come to their assistance.

And as the temporary hospitals filled up, the confusion and scenes of horror only increased. Men died from neglect under the very eyes of those who could have saved them had appliances existed. Then, when medical comforts did arrive, the number of attendants was so small that their utmost efforts did not avail to help every one.

Dunant persuaded the villagers to come to the rescue, and many of them took pity on the wounded French and nursed them in their cottages. But one night there was an alarm that the Austrians were returning, and instantly all the peasants cast

out the French from their houses, many of whom perished, because their hosts feared reprisals from the Austrian army. Some of the women did noble work in the hospitals, but their help was clumsy, and many of them sickened at such painful sights and could not be persuaded to return. "Ah!" cries Dunant, "how inestimable would have been a hundred voluntary hospital orderlies and nurses distributed among the villages of Lombardy, trained and well-qualified for such work! They would have rallied round them the scattered forces of willing hands who needed guidance, for not only had those capable of teaching no time to teach, but the efforts of those who wished to help often proved fruitless because they lacked knowledge and skill."

His book first gives a vivid description of all he saw, the terrible plight of the wounded, the crowded hospitals and the over-worked staff, and then proceeds to urge the formation, in all the leading countries of Europe, of a voluntary service of workers trained to supplement the regular medical service in time of war. This proposition to form, "during times of peace and tranquillity, relief societies, whose aim should be to help the wounded in time of war, by means of volunteers, zealous, devoted and well qualified for such work," was duly considered by the Geneva Society of Public Utility, of which Dunant was a member, in 1863, and a commission of five was nominated, with General Dufour as its President and Dunant as Secretary, to consider the question.

This Commission called a conference in Geneva in October of the same year, and invited representatives of every European power to take part in the discussion.

Meanwhile events in America had proved to all the world the immense value of charitable assistance, properly administered, in war when skilfully and liberally supplied. Civil war had broken out in 1861, and while volunteers arose in hundreds of thousands to join the colours, other men and many women, in several districts, formed themselves into bands of workers for collecting and distributing supplies for the relief of the sick and wounded. Hardly had these societies been formed, in the teeth of the most determined opposition at headquarters, when their value became patent to all.

Thus, after the battle of Antietam in September, 1862, more than 12,000 wounded had to be dealt with, and the supplies of the medical authorities were only one tenth of what was needed to save a vast amount of unnecessary suffering. Fortunately the relief societies had arranged for relays of trains to come to the army every day with supplies, and, thanks to their help, the most pressing needs of the wounded were relieved. Besides chloroform, instruments, bandages, etc., supplied by the Commission, an auxiliary relief corps of men and women was formed to tend the wounded in the hospitals, others established food stations on the railway to feed the wounded on their way down to the base hospitals, and all these people toiled unweariedly through months of the hardest work, and while some were captured and kept as prisoners others died at their posts.

"Thank God, here comes the Sanitary Commission; now we shall be able to do something," was the constant cry of the army surgeon. Could any devoted worker seek higher praise than that?

Whilst the American Sanitary Commission was doing this splendid work across the Atlantic, the Conference called at Geneva met in October, 1863, and was attended by thirty-six members; fourteen Governments, including those of Russia, Sweden, Italy, Prussia, France, Austria, Spain and Great Britain were officially represented. This Conference sat for four days, and adopted the resolution that committees should be formed in every country to supplement, by preparation in time of peace, the regular hospital service of the army in time of war. Some further suggestions were not supported, but before separating, the delegates recorded a strong desire to appeal to the Governments of all civilised nations to agree to the neutrality of the sick and wounded, the hospitals sheltering them, the staff attending on them, and any inhabitants of the country who were assisting them; they also urged that a common recognisable sign should be adopted by all the medical staff, and that a common flag should fly over every military hospital.

To consider these questions the International Congress of 1864 was called at Geneva, that place being chosen as a suitable, non-contentious site belonging to a small independent nation with no standing army. The invitation was accepted by sixteen powers, and the Congress opened with twenty-five representatives, who deliberated for nearly a fortnight before they signed the Convention.

Before this date it had been the practice to consider the sick and wounded as lawful prizes in war, to be captured if possible, as well as the medical staff, the ambulance transport and equipment. The Congress had to determine whether these men and material might not, in common humanity, be treated as neutrals, and it was determined that they should in future be safe from attack or disturbance.

Next, it was considered advisable, in order to secure the aid of the civil inhabitants near the scene of battle, to grant them, not only protection, but a dispensation from the billetting of troops if they would receive the wounded into their houses.

The disabled were not, in future, to be considered as prisoners of war.

Finally, the distinctive Red Cross on the white ground was adopted as the flag to fly over military hospitals, to be painted on ambulance waggons, and to be worn as a badge on the arm of the medical and nursing staff. This emblem was decided on to show the Christian principles underlying the Convention, and as a delicate compliment to the Swiss Government, the design being similar to their national flag, with the colours reversed.

The practical appeal for willing hands to come to the help of the sick and wounded in war met with an instant response from almost every country of Europe. England, Holland, and Russia held aloof for a short time, but here also the greatest interest was taken in the movement; nor, as we shall see, were we behindhand when the time came for carrying into effect Red Cross sentiments.

The short winter campaign of Schleswig-Holstein, when the Prussians and Austrians invaded Denmark, was fought the same year as the Convention was signed, and Red Cross workers belonging to the invading forces found ample opportunity of supplementing the regular army medical service. As in former wars, the hospitals were filled to overflowing after several engagements had taken place, and the Red Cross Society appealed to the inhabitants of the district to take some of the sick and wounded into private houses. This was done, resulting in the immediate decline of the death-rate. Individual soldiers were helped in various ways, and this, the first organised attempt at Red Cross work, proved the value of such assistance.

When the war came to an end the Berlin society formed itself into a permanent institution, and other countries followed suit, each conforming to the principles laid down by the Geneva Convention.

In 1866 the two conquerors, Prussia and Austria, went to war with each other, and in the seven weeks' campaign the Red Cross Society did splendid work, rendering assistance to friends and foes alike, in the true spirit of the Convention. After the battle of Königgrätz 40,000 wounded were lying on the battlefield, and it became an impossible task for the regular army medical service to transport, house, or attend to such numbers. For three days and nights the bearers searched the field, relieving the wounded where they found them, but hundreds died before help came. The service of the volunteer civil surgeons proved to be of inestimable use in assisting the army surgeons, and the Society conveyed the overflow of wounded to private houses within reach, placed every wounded man on a good bed and supplied him with medical comforts.

So far the Red Cross societies had assisted the wounded of those countries from which the particular societies sprang, and Germany, being primarily engaged in the wars, had taken the lead. England had had no part in the work. But the Geneva Convention embraced the possibility of assistance being given, under particular circumstances, to the sick and wounded of foreign countries.

Dr. T. Longmore, Professor of Military Surgery, had urged in 1866 the importance of Red Cross work in England, and the possibility of forming a society for the purpose was constantly in the minds of Mr. (now Sir John) Furley, Colonel Loyd-Lindsay (later Lord Wantage), and others. Sir John Furley had attended the International Conference of Red Cross Societies in Berlin in 1868, and although he had to admit that no such society as yet existed in his own country, he pledged his word that Great Britain would not be behindhand in such work should the necessity arise. Within two years this pledge was put to the proof, when Red Cross work in England was started on behalf of two foreign armies on foreign soil.

War between Germany and France was declared suddenly in July, 1870. A week later Sir John Furley called on Colonel Loyd-Lindsay, and proposed the formation of a British Red Cross Society. A letter was written to 'The Times,' appealing for

funds for the relief of the sick and wounded, and it became at once the main work of the Society to direct the national outburst of sympathy which answered the appeal. This earliest Red Cross movement was started under the name of the National Aid Society for the sick and wounded in war, with Queen Victoria as patron. Provincial committees were formed all over the country, unbounded generosity was evoked, money and gifts poured in.

Within two weeks £30,000 had been subscribed. It was no easy task to improvise everything that was necessary for the proper distribution of such funds, and Sir John Furley went to Geneva to confer with the International Committee sitting there. The house taken in St. Martin's-in-the-Fields for offices for the Society soon proved too small for the vast stores of every kind of material which were contributed for the relief of the wounded, and two houses opposite were taken, then the churchyard was tented over, and finally the large vaults below the church were used, besides a portion of an adjoining workhouse. Within three weeks forty British surgeons had joined the opposing forces, and were hard at work in the hospitals and at the front. By the middle of September the number of surgeons had risen to sixty-two, and sixteen ladies were acting as nurses. Large depôts of stores were formed at different places on the Continent, and branch hospitals were instituted under British management, with funds from home. Let me quote an incident from Sir John Furley's own story, just after the battle of Sedan :

"We passed through the still smoking ruins of the village of Bazeilles . . . there was not a house or a garden that was not utterly destroyed. . . . In the mairie of Balan we found Dr. P. Frank and Mr. Blewitt hard at work, and as I watched them from bed to bed, I felt that, if our Society had nothing else to show except what was here exhibited, we might well have reason to be grateful. The building bore many marks of the perils to which these gentlemen and those who aided them . . . a dyer of Balan and the two daughters of the village schoolmaster, who managed the kitchen, had been exposed on the eventful day of Sedan. The windows were shattered by bullets, which, passing on, had left their traces on walls and furniture; here and there, too, heavier metal had penetrated. The surgeons and their assistants had been obliged to work amongst the patients on their knees in order to keep their heads below the window-sills; one lady, who had forgotten the necessity of doing so, had received a bullet through the hair just above the nape of the neck."

The National Aid Society obtained permission, at first refused, to take chloroform to the hospitals at Metz during the siege; it sent out twelve ambulance waggons, hundreds of iron bedsteads, and food, medicines and warm clothing of every description, besides establishing railway-station soup-kitchens to refresh the wounded on their journeys.

Mrs. Loyd-Lindsay, who was actively engaged in receiving and dealing out stores at the Committee-rooms in London, wrote: "Every letter that comes from abroad begs for more things, more instruments, more chloroform, more morphia; the want of these things conveys an awful idea of the extent of pain and suffering." Colonel Loyd-Lindsay went to Paris, and was admitted under the flag of truce during the siege, in order personally to superintend the distribution of medical comforts and to find out the special needs of the hospitals. "I went through most of the hospitals both at Versailles and Paris," he wrote. "At Versailles the wounded were all lying in the magnificent galleries, with the great pictures of the French victories around them. It sounds luxurious, but though the rooms are large and lofty, the ventilation is bad. The hospitals on the whole seem moderately well managed, depending entirely on voluntary aid societies for the supply of everything in the shape of extra comforts, such as changes of clothes, warm flannels, good food, drinks, etc. Our money and our supplies will, therefore, be of the greatest use and comfort. In Paris the French seem to have done their best, but they are badly off for surgeons and for many other necessaries; the nurses are chiefly men-orderlies, but a great many women and ladies give their voluntary aid."

During the winter it was found that French prisoners in Germany and German prisoners in France were in a pitiable condition from a total lack of proper clothing, and the Society was enabled to distribute warm clothes and other small comforts to them.

The work the National Aid Society did during the war was, to a great extent, experimental; there had been no preparation and no experience, and to this extent it fell short of the ideals set by the Geneva Convention. But its work was carried out by untiring voluntary helpers, both at home in organising and at the front in actively assisting. It sprang into being at a moment of national emotion, but it was built on a more solid foundation than that.

It became evident to many that there was a real need for a permanently established society for the relief of the sick and wounded in war, and that England must not leave to others that noble task. The work of preparation, of the training in first aid to the injured, which has since become a household word in this country, was, however, not at first undertaken by the National Aid Society. The Council considered it too great an enterprise, and the Order of St. John of Jerusalem in England undertook the training of voluntary workers in First Aid in 1878. Leaving aside at first the questions relating to hospital assistance to be given in time of war, they found a sufficient scope for their energy in training members of the public in the assistance which everyone ought to be capable of giving in the accidents which occur in the daily life of the civil community and in home nursing. In due time the War Office became aware of the fact that such voluntary trained workers might become a valuable supplementary corps for service in the field, and the organisation of workers was carried on in response to this desire.

After the International Conference of Red Cross Societies held in Vienna in 1897, the Secretary of State for War, the Marquess of Lansdowne, invited representatives of the National Aid Society, the St. John Ambulance Association and the Army Nursing Reserve to meet him in an informal conference to discuss the advantages of bringing volunteer aid societies into touch with the Army Medical Department, in order to make these societies of acknowledged use in supplementing the regular medical service in time of war. As a consequence of this meeting the Central British Red Cross Council was formed and was officially recognised by the War Office, which appointed its own representatives to it, whilst the Princess of Wales, now Queen Alexandra, graciously consented to become President of the Council.

The British Red Cross Society is now an established institution in this country. Sir John Furley, the *doyen* of the movement, who has been interested in its work since its origin, and who has attended, during the last forty years, seven International Conferences on the subject in Europe and Washington, maintains, in his paper recently read at Washington, that, "The ideal Red Cross Society is a national institution which keeps only one object in view, and that is, the alleviation of the sufferings of the sick and wounded sailors and soldiers in war. But it should be the centre of various other philanthropic societies, each performing an important work in peace, and yet so constituted that . . . they can be depended on to give substantial aid to the Royal Army Medical Corps in time of war."

Of work done by the National Aid Society in some wars subsequent to 1871 I hope to speak to you in my next lecture.

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November 25th, 1912.

We have received from Burroughs Wellcome & Co. a bottle of "Paroleine" Spray Compound; this contains suitable proportions of menthol, chlorbutol and "Eucalyptia" in a basis of "Paroleine," the whole forming an excellently designed preparation to be applied as a spray to the nose or throat. The combination is a well-tried one, and has been found exceedingly useful for its sedative, analgesic and antiseptic effects in catarrhal conditions of the respiratory tract generally. The use of chlorbutol as the anæsthetic ingredient obviates the danger of inducing a drug habit, which is always present with sprays containing cocaine. "Paroleine" Spray Compound is free from irritating action on the mucous membranes.

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TWO LECTURES
ON
CRANIO-CEREBRAL SURGERY.

Delivered at St. Bartholomew's Hospital.

By L. BATHE RAWLING, M.B., B.C., F.R.C.S.,
Surgeon with Charge of Out-patients to the Hospital.

LECTURE I.

GENTLEMEN,—With respect to the two lectures which I have the honour of delivering to you on cranio-cerebral surgery, I propose next time to deal with the subject of tumours of the brain and their surgical aspect. To-day, I shall go over with you some facts in connection with fractures of the base of the skull. In these days of rapid transit by motor car, aeroplane and the like, you will find that fractures of the base of the skull are showing an ever-increasing frequency of occurrence, and on that account, and by reason of the fact that surgical procedures are being advocated with ever-increasing persistency and with considerable success, the subject will probably be of some interest to you.

You will agree with me, first of all, when I say that the diagnosis of a fracture of the base of the skull is dependent to a very large extent on certain visible external hæmorrhages, on the discharge of cerebro-spinal fluid from the ear, nose and mouth, and on certain nerve palsies. Still, in spite of the fact that these clinical symptoms are of great value to you from the point of view of diagnosis, they are not of much value from the standpoint of prognosis. Whether a patient is going to live or to die hinges in the main on the question of associated lesions of the brain, venous sinuses, and meningeal arteries.

I propose first of all to go into certain points with regard to these symptoms, illustrating the various basic fractures with which they are associated. Then I will go over one or two points with regard to intra-cranial lesions—lacerations of

the brain, middle meningeal hæmorrhage, and hæmorrhage between the dura mater and the brain. Finally I shall go on to deal with certain points in connection with the treatment of fracture of the base of the skull, more especially the treatment of what we may call the "average" case.

Here is a picture of the base of the skull showing the various fossæ. Next you will see the same base under transillumination. You will at once see that there are in this base certain specially weak areas, the orbital plates of the frontal bone, the cribriform plate of the ethmoid, the middle meningeal territory, as it is termed, and the weak portions of the cerebellar fossa. It is perfectly obvious that these three portions of the base are more liable to fracture than any other part of the skull. Here you see plans which illustrate what I may call the preferential lines of basic fractures, that is to say the lines along which basal fractures tend to traverse if they have the chance. Basic fractures follow the weaker lines of the base; and without going into any great detail there are a few points I would illustrate. You will notice that all fractures of the anterior fossa either pass across the orbital plate of the frontal bone, or the cribriform plate of the ethmoid, and thus you have a satisfactory explanation of the various orbital hæmorrhages that are observed—subconjunctival hæmorrhage, palpebral hæmorrhage, proptosis and the like, whilst, through these cribriform plate fractures, nasal hæmorrhage is accounted for. Let us now take the various middle fossa fractures. Here, for instance is a fracture running across the middle fossa. They all tend to implicate this central portion of the basis cranii, the sphenoidal sinus. This region assists in the formation of the roof of the naso-pharynx, and all fractures involving the sphenoidal body lead to hæmorrhage from the nose and mouth. Furthermore, on either side of the basi-sphenoid there are the two cavernous sinuses, and in many cases the sphenoidal region is so severely injured and comminuted that the cavernous sinus shares in the lesion, and thus you have an explanation of those very severe cases of hæmorrhage into the nose and mouth which are commonly present when the fracture involves this region. And there is another point to which I would like to draw your attention in connection with what I have named the "typical" fracture of the base of the skull, so-called by reason of its frequency of occurence and constancy of direction. It is a fracture which runs along the front part of the petrous bone, and whether it pursues its original direction into the opposite anterior fossa, terminates in the sphenoidal region or involves the opposite middle fossa it matters not. In any case, the fracture runs along the front part of the body of the petrous bone, between the petrous and the wing of the sphenoid. It is in this class of case that you find hæmorrhage from the ear and mouth, and certain cerebral palsies. There is hæmorrhage from the ear by reason of the fact, as I shall show you presently, that these fractures all involve the tegmen of the middle ear and the roof of the external ear, incidentally lacerating the membrana tympani, and free bleeding from the mouth by reason of the fact that they all involve the body of the sphenoid. And there is also paralysis or paresis of the seventh nerve insomuch as the fracture passes immediately in front of the geniculate ganglion of the facial nerve. I will indicate the relation of the facial nerve to this "typical" fracture. From the internal auditory meatus the nerve passes forwards and outwards, then backwards, the geniculate ganglion being situated at the angle between these two lines. This ganglion is compressed by blood or by bone in most middle fossa fractures. The facial paresis or paralysis which results is, however, seldom permanent. There is another class of fracture of the petrous bone to which I will now direct your attention. It is a fracture originating in the posterior fossa, passing across the petrous bone in the antero-posterior direction and severing the facial nerve into two. It is in this class of fracture that you find complete and permanent paralysis of the facial nerve.

Here are some photographs of basic fractures illustrating some of these points. Here is a fracture running from the posterior fossa into the anterior fossa. It has comminuted the sphenoidal body, and probably torn the cavernous sinus, and passing inwards it has produced an extensive lesion of the cribriform plate of the ethmoid. This patient would have had severe hæmorrhage from the nose and mouth. Here is another picture of a fracture of the anterior fossa which passed backwards across the orbital plate of the frontal bone and terminated in the cavernous region. There are some slight side comminutions into the cribriform plate. This patient had severe hæmorrhages in and around

the orbit, bleeding from the nose and profuse hæmorrhage into his naso-pharynx, unusually severe by reason of the fact that the cavernous sinus was extensively lacerated. Here is a picture illustrating the typical fracture of the middle fossa. It originates in the aural region and passes inwards along the front part of the petrous bone, terminating in the region of the sphenoidal body. Here is another illustrating a similar condition. This white mark which you see indicates the angle of junction between the two parts of the facial nerve. You see that the fracture passes along the front part of the petrous bone, between it and the great wing of the sphenoid, immediately in front of the geniculate ganglion, terminating finally in the sphenoidal region. Here is another. They are all more or less the same. This particular one was associated with a secondary fracture across the petrous, in the antero-posterior direction. Here is one which is rather interesting because it illustrates a united fracture of the "typical" middle fossa type. It was discovered accidentally in the post-mortem room, and it shows an old basic fracture from which the patient recovered. In other respects it follows the usual direction. This next picture is perhaps a little difficult to understand, but it illustrates the more exact course of this typical fracture. It runs from one ear to the other, not in a straight line but forwards to the sphenoid body, and then backwards to the opposite ear. This is the anterior aspect of the posterior fragment. You will observe both external and middle ears, and here, in the middle line, are the two sphenoidal sinuses. This case would have been admitted with severe hæmorrhage from the naso-pharnyx and with a lesser degree of bleeding from both ears. I would impress the fact that when you see a patient with bleeding from both ears, especially when associated with hæmorrhage from the mouth as well, you can be practically certain that you have to deal with this particular type of fracture. Here is the petrous bone in a case of this nature showing the conditions a little more closely. The next illustration shows that other variety of fracture which I have previously referred to, that in which the petrous is cut across in the antero-posterior direction. The fracture originates in the posterior fossa and passes forwards to the post-condyloid region of the foramen magnum on the one side, starts again on the opposite side of that foramen, and then cuts across the petrous bone in the antero-posterior direction. You can see that this antero-posterior fracture, if sufficiently extensive, will sever the seventh nerve and divide the auditory apparatus into two parts, and so in this particular variety of fracture there is complete facial paralysis and permanent deafness. Here is a skull in which the inner portion of the petrous bone has been thrown forward so that you can see its relation to the auditory apparatus and to the course of the facial nerve. Next we have other petrous bones showing the same antero-posterior fracture. The next slide is to show you the outer part of the petrous bone in a case of antero-posterior fracture. It is meant to illustrate the rather important clinical fact that, in this class of basic fracture, the drum is uninjured. The drum remains absolutely intact, and consequently, although the patient exhibits paralysis of the face muscles and is stone deaf, there is no bleeding from the ear.

Here is a section of the skull, anterior-posterior, and next to it you see the same skull as seen on transillumination. This picture illustrates very well the extraordinary weakness of the skull in the temporal and temporo-parietal regions, in what one may call the middle meningeal territory. Here is the middle meningeal artery, and you can see its branches passing in fairly definite directions. It is obvious therefore that any blows received on the side of the head are very liable to injure the middle meningeal artery, and more especially those branches which run in an osseous channel. These next two pictures illustrate a point which is of considerable clinical value. In your practice you must frequently meet with cases in which the bleeding from the ear is exceedingly profuse. This particular specimen was obtained from a patient under my care, my attention being at once drawn to the possible source of profuse aural bleedings. The fracture originated in the aural region, passing upwards to the vault and downwards to the base. On tracing it inwards you will see that it comminutes extensively the roof of the middle and external ears. This groove which you can see is caused by the posterior branch of the middle meningeal artery, which vessel was torn with a resultant extensive hæmorrhage between the dura mater and the bone—immediately above the tegment tympani. The blood was forced downwards into the middle and external ears and came out through the ex-

ternal auditory meatus. This relation of a middle meningeal hæmorrhage to a fracture of the middle fossa was, as I have said, accidentally discovered. But the appearance of the skull so impressed itself upon me that I investigated a considerable number of other cases afterwards to see whether the profuseness of the hæmorrhage from the ear had any relation to the degree of middle meningeal hæmorrhage. And I found the more profuse the hæmorrhage from the ear the greater was the chance that there was an extra-dural clot from a torn middle meningeal artery. The important fact to be observed in connection with this is that the worst treatment you can carry out is to plug the ear, as it only leads to the imprisonment of the hæmorrhage and the increase of the already existent brain compression. And a still more important fact is that the sooner you operate in cases of this sort, evacuating the middle meningeal clot and ligaturing the middle meningeal artery or its branch, the better for the patient. Here is a picture of an ordinary middle meningeal hæmorrhage. The clot is more or less lens-like in shape, and it is exercising marked compression on the underlying cortex. Time will not permit me to enter into the typical clinical symptoms of middle meningeal hæmorrhage, but I would point out to you that any patient who comes under your care conscious and who gradually loses his senses within the next few hours, more especially if he also exhibits other symptoms of cerebral compression, that patient has almost certainly a clot inside his skull, either between the dura mater and the bone or between the dura mater and brain. In the former instance the hæmorrhage is almost necessarily dependent on torn meningeal vessels; in the latter the clot results from lacerated cortical vessels. In either case, the sooner that clot is evacuated the better for the patient. In the event of hæmorrhage from the middle meningeal artery, trephining is conducted over the middle meningeal territory, the area I have previously mentioned, preferably where the line of a fracture crosses the line of the middle meningeal artery. In cases of subdural hæmorrhage, on the other hand, the localising features are rather obscure. There is, however, one important clinical fact on which I would lay stress. In the case of extra-dural hæmorrhage (from the middle meningeal artery) there is usually some lucid interval between the time of the accident and the onset of compression symtoms, lasting perhaps only a few minutes, perhaps two or three hours or more. In extensive subdural extravasations the patients, so far as my experience goes, never recover complete consciousness after the accident, but they show an ever-increasing depth of compression from the beginning to the end of the case.

From the point of view of treatment of these cases I do not want to lay too much stress on the temperature chart. I would, however, rather take that as a valuable guide to your treatment, for if there is any single symptom in your head cases which is of greater value than any other, it is the alterations in temperature. This chart which I show you was framed from a large number of cases of head injury, and every case that comes under your care will fall quite readily into one or other of these groups. Here is a chart which exemplifies those cases of head injury in which the temperature rises progressively from beginning to end. You must have met several of these cases in your practical experience. The patient comes in with subnormal temperature—the period of shock. Shortly afterwards the temperature begins to rise; it rises progressively, and in twenty-four to forty-eight hours, unless something is done, the patient is dead. All these cases which show a progressive rise of temperature die unless operative measures are carried out. Another group is that in which the patient is admitted with subnormal temperature, and in spite of everything you can do it remains subnormal, and this patient also dies invariably within forty-eight hours or so unless radical measures are carried out.

There is a third class in which the temperature, at first subnormal, rises to, and remains at, normal. There need be no great anxiety about him. Another class of case is where the temperature rises to 101° or 102° F. and then halts, and, as it were, marks time. If you watch this case you will find that if the temperature begins to go up the patient will probably die, whereas, on the other hand, if the temperature begins to come down, the patient almost invariably recovers. So by observing the temperature of these head cases you can in the majority of cases, come to very definite conclusions, from that smyptom alone, as to what will take place. And added to that, I think you can form

a very correct idea as to what is the appropriate treatment. Take first this case: here is a patient suffering obviously from concussion—what we call cerebral shock—with a persistent subnormal temperature, and this patient, unless by your measures he is brought out of that condition, will die. Therefore you must go in for heroic measures, and you ought to treat these cases in exactly the same way as you treat a case of ordinary surgical shock. If you have a patient of this sort brought into the hospital more or less moribund, in a collapsed condition, with quick feeble pulse and subnormal temperature, the sooner he is infused *per rectum* the better for the patient. If he is excessively bad you can infuse him by the intra-venous method. Under other circumstances I think it is wiser to utilise the slower but safer method of rectal infusion. The patient's condition of shock is probably dependent on a brain lesion, laceration of the brain with subdural bleeding, and if you infuse *per rectum*, putting in two or three pints of saline solution, you will almost certainly raise the blood-pressure, but you will at the time same tend to induce a recurrence of the intra-cranial bleeding. That is the danger which you must bear in mind. Still, on the other hand, you must understand that the patient will die unless you do something. If you infuse this patient *per rectum* and bring him out of his condition of shock, as is verified by the rising temperature and increasing blood-pressure, he falls automatically into group 1, that group which shows a steady and progressive rise of temperature. This class of patient, provided that he is not too far gone, almost invariably demands operative treatment. They all have laceration of the brain, with subdural hæmorrhage, or else they are suffering from an extra-dural middle meningeal bleeding, both of which conditions, by reason of the resultant brain compression, require energetic operative measures. The operative details vary, of course, according to the symptoms evinced by the patient. If there is a middle meningeal hæmorrhage you operate on the territory of the middle meningeal artery, and if there is bleeding from a torn venous sinus you explore that region first.

I think these temperature charts will help you from the point of view of treatment, for, if we take another example, the indications are fairly obvious. The patient who has a normal temperature, or who shows but slight elevation of temperature, is going to get well. He requires no special operative treatment. He will get well whatever you do, and therefore nothing radical is required You only need keep him quiet, nothing more. Or the other hand, this patient with a "hovering" temperature, rising from the subnormal to about 101° or 102° F., and there marking time, he is the type of patient upon whom I think venesection is of the greatest possible use. I am not quite sure whether venesection is based upon sound scientific principles—I mean scientific in relation to the theories which we have in respect to concussion and compression—but from a practical point of view venesection is of very great use in cases of this sort. When the patient has this so-called hovering temperature, and when he is half-way between the concious stage and the stage of compression, merely recognising his surroundings to a limited extent, then if you carry out venesection I think you will obtain fairly satisfactory results. In many cases the adoption of venesection will turn the tide in favour of the patient. Venesection is to be carried out in the usual way, from the median basilic or cephalic veins. The amount of blood to be withdrawn is dependent on the condition of the patient. If he is full-blooded and apoplectic you can take a pint away, but under ordinary circumstances I think half a pint of blood is sufficient. In any case, while the bleeding is going on, keep a sharp eye on the patient, and as soon as his pulse begins to increase in frequency and becomes softer, it is time to stop. The process can be repeated later if required.

If you are operating on cases of fracture of the skull, for brain lesions or for tumours of the brain, it is necessary to reduce all possible hæmorrhage to an absolute minimum. Scalp bleeding can be obviated by utilising the scalp tourniquet. This appliance was first recommended by Harvey Cushing. This picture shows the type which I am accustomed to utilise. The head of the patient is first of all covered with gauze, drawn over the head as is shown in this picture, and maintained in position by means of the tourniquet. The tourniquet which I use consists of rubber, and it is tightened up in such a way that all the circulation from the occipital, temporal and other scalp vessels is efficiently controlled. A piece of tape passing over the head prevents the tourniquet from sliding over the eyes. The incision down to the bone can

be carried out in one single sweep, and if the tourniquet is working correctly the amount of hæmorrhage is absolutely *nil*. That is a point of enormous value in consideration of the time required for many of these head operations. You will find it is generally considered that the degree of shock from head operations is very excessive. My own experience shows that this is quite erroneous, the degree of shock being proportionate to the amount of blood which is lost. If you turn down a flap in the old-fashioned way you know how serious the bleeding is. On both the convex and the concave sides of the flap the bleeding is profuse, both during the formation of the flap and during the efforts carried out for its control. The amount of blood lost by the patient during this preliminary stage is very great. I have no doubt that this blood loss exercises a very strong contributory influence on those severe cases of shock which were observed in connection with head operations some years ago. Since Harvey Cushing instituted and carried out this means whereby scalp bleeding is prevented, we find that the degree of shock from which these patients suffer is very much less than it was before. The next picture shows you a back view of the tourniquet in position. You will observe that it occupies such a position that it can be utilised in many operations on the head. In operations in the temporal region—such as Gasserian ganglion removal—other procedures have to be adopted whereby to lessen the degree of bleeding.

If you have to deal with subdural hæmorrhages, this next picture will show you the best way in which to evacuate the clot. This patient suffered from hæmorrhage beneath the dura mater, the clot extending nearly up to the middle line of the skull and down to the base. The bone was trephined and the diagnosis confirmed. Then the dura mater was opened in the region of the trephine hole in a crucial manner, the extent of the clot in the downward direction being investigated with a probe. Having obtained this information, a narrow channel was cut in the bone to the lowermost limit of the clot, and the dura mater slit up all the way. Then the clot is evacuated. It is very difficult to evacuate the clot completely. You can get away about two thirds, and, in most cases, you must there rest content. In all probability the removal of that quantity will suffice. You can see in this figure the way in which the dura mater is sewn up afterwards. A drainage-tube was inserted beneath the dura mater and brought out through the scalp flap, the rest of the dura mater being sewn up, first by the cross union of these four dural flaps, and then by a few sutures which brought together the rest of the dural incision. Cases of subdural hæmorrhage on the whole do fairly well. But I do not think you can often absolutely cure your patient. Many of them suffer from some of the remote effects of head injury, but still you can often save your patient's life, and that is sufficient for the time being.

I want now to direct your attention to the treatment of the average case of fractured base. I think you will agree with me when I say that it is but a small proportion of cases of fracture of the base which give typical localising symptoms, such, for instance, as point to a middle meningeal hæmorrhage, or to a localised subdural hæmorrhage. The average case of fractured base is diagnosed by certain external symptoms, such as hæmorrhage from the ear, nose, mouth, etc., and when such a patient remains unconscious for a prolonged period of time or shows evidence of compression, we know from experience that the majority of them have laceration of the brain with hæmorrhage beneath the dura mater. You can, in the milder types of compression, venesect the patient, with fairly satisfactory results. In a very large proportion of cases, however, you will find that much more radical measures are necessitated. Those measures are called measures of decompression. There are two areas over which you can decompress the brain—the cerebellar region and the temporal region. I will deal with the cerebellar region first because I have not formed a high opinion of cerebellar decompression. I have tried it on many occasions, but I have never found any case show more than temporary improvement in consequence of it. It is obvious, inasmuch as the vital centres are grouped in the subtentorial region, that cerebellar decompression ought to relieve the compression with great rapidity, and with greater efficiency than when a decompression operation is carried out over any other part of the brain. And such is the case. I used to carry out cerebellar decompression, but I found that, although some cases improved greatly for the time being, yet that I never obtained any definite permanent

improvement. In fact, I think I can say I never saved a patient's life by cerebellar decompression. That is my experience. Then people begin to search round for some other region over which one could decompress, paying special attention to those regions of the brain which are regarded as "silent" areas—areas possessing no important known function. You do not want to decompress a patient over his Rolandic area. If that particular part of the brain extrudes through the gap in the bone he will have spastic paralysis of the extremities on the opposite side. Here is a picture which shows you at once the area over which you can carry out a satisfactory decompression—the temporo-sphenoidal lobe. This region is chosen because it is of practically little value in the sense that you can allow the temporo-sphenoidal lobe to herniate out through any hole which you may make in the skull and no untoward symptoms of any important character result. It is for this reason that I advocate that decompressions should be carried out over the temporo-sphenoidal lobe.

Here is another picture to show the various areas of the brain. You will notice that any decompression which is carried out over the temporo-sphenoidal region must be conducted low down near the basis cranii; you must not decompress the patient so that any portions of the lower Rolandic areas are included in that part of the brain substance which is going to protrude through the opening. On the left side of the skull this point is of even greater importance, by reason of the presence of Broca's speech area.

I want now to enter into further detail with regard to the intermusculo-temporal method of decompression which was first carried out and described by Harvey Cushing at Johns Hopkins Hospital. He chose the temporal region, and he did so for several reasons. One was because the operation was carried out over a silent area of the brain, the temporo-sphenoidal lobe. The second reason was because the operation was carried out over the middle meningeal territory, that is to say over that part of the skull where the middle meningeal arteries ramify; for he had proved—and my experience was exactly the same—that middle meningeal hæmorrhages are exceedingly common. You do not often find a middle meningeal hæmorrhage which yields those typical symptoms which you find mentioned in your text-books. But if you operate on your cases, and see others in the post-mortem room, you will find that hæmorrhages between the dura mater and the bone are of very great frequency, and this in spite of the fact that they may lead to none of those very definite clinical symptoms of which you read. An exploration of this middle meningeal territory often reveals an extra-dural hæmorrhage which would otherwise have been unsuspected. Another reason is that the operation is conducted underneath the fibres of the temporal muscle—it is a splitting operation. The incision starts above and behind the external angular frontal process, curves along the temporal crest and passes downwards to the top of the ear or even a little lower. The temporal fascia is then slit up with scissors, immediately below the line of the temporal crest, and turned down. That exposes the temporal muscle, the fibres of which are split. Then the bone is trephined. As regards trephining there are all sorts of trephines, electric and otherwise. But for my own part I would advise you to remain content with the ordinary hand-trephine, so long as you get one which is well and correctly made. The hand-trephine may be slow, and may be regarded by some people as cumbersome, but I have tried all methods and I have always come back to the old hand-trephine, for the reason that it is completely under one's control, and entirely safe from the point of view of the patient. No motor trephine of any sort can possibly have the same degree of safety as an ordinary hand-trephine. The bone is trephined in the middle of the exposed area, and then, retracting the temporal muscle widely on either side, you cut away the bone backwards and forwards and a little upwards. I say "a little upwards," because although this picture illustrates the operation on the right side of the brain, the operation may be conducted on the opposite side, and you must avoid any possibility of including the motor speech area of Broca. So the bone is cut away freely, and in this way you open up a ready means of access to the temporal fossa of the skull. Next you incise the dura mater in a crucial manner. And that reminds me that I have often been asked this question: Is it necessary in decompression operations to open the dura mater? Of course, it is absolutely necessary. The removal of the bone alone has practically no effect worth speaking of with regard to intra-dural tension. You must open the dura mater freely, and preferably by means of

a crucial incision. The dural flaps are allowed to retract, and any underlying clot or brain matter under high tension would at once tend to protrude through this dural opening. After that there is no need to do any more except to approximate the fibres of the temporal muscle and sew them together. You know the degree of hernial protrusion varies, but, if the patient recovers, the brain receives adequate protection from this muscular covering. The temporal fascia is sewn up, and finally the skin approximated. One point about this temporal fascia: This illustration was taken from the cadaver; in actual life you will not always find yourself able to sew up accurately the temporal fascia, because it retracts considerably, and you have to do the best you can. The temporal muscle can always be united, but the fascia you sew up as well as circumstances permit, one layer protecting the other. It is a splitting or gridiron operation, similar to that carried out for the removal of the appendix. If you like you can insert a drainage-tube beneath the temporo-sphenoidal lobe, which would have been exposed by this incision of dura mater. You bring out your drainage-tube through the flap. But I am a little doubtful whether it is advisable to drain. I would lay a good deal of stress upon this particular operation of Cushing's, because, when carried out in appropriate cases, it produces very satisfactory results.

If you see and treat many of these head cases, you will frequently be disappointed with your results. Many patients have very severe lesions of the brain, and some of them cannot be expected to recover, whatever you do. Still, you have to make an attempt. In a certain proportion operative measures will be of considerable service and save the patient's life. There is one other advantage claimed for this intermusculo-temporal operation. The anterior pole of the temporo-sphenoidal lobe is exposed, a part of the brain which is more often lacerated than any other. Therefore, you will see in general that Cushing's operation is founded on accurate anatomical and scientific principles.

Here are some specimens illustrating some of the points to which I have alluded. Here is a fracture of a child's skull, showing extensive damage to the anterior fossa, more especially in the region of the cribriform plate. There was very severe hæmorrhage from the nose and mouth. Here is a fracture of the posterior fossa, and here is another fracture of the typical type running along the front part of the petrous bone. This next is the skull of a policeman, who committed suicide with a pistol. It possesses considerable interest in connection with the treatment of bullet-wounds of the skull. The bullet entered in the right temporal region, injured both orbital plates, comminuted but failed to perforate the left temporal fossa, and finally fell back into the right lateral ventricle, and at the post-mortem it was found in the descending cornu of that ventricle. The point about bullet-wounds is that unless the bullet is found readily, either at the entrance or at the site of attempted exit, you should not do anything further except after full X-ray investigation, the immediate application of which is not always possible. The next specimen shows a middle meningeal hæmorrhage of the typical type. In this case, in the post-mortem room, an old blood-clot fell out as soon as the dura was opened. The patient suffered from headaches and fits, and here is the cause. If a hole had been made in the skull during life the whole of that clot could have been removed. The next specimen shows the ordinary type of laceration of the brain. Note that parts mainly involved are the temporo-sphenoidal pole, and the anterior and inferior aspects of the frontal lobe.

In conclusion, I would express a hope that these few remarks may be of some assistance to you in the treatment of those injuries of the head which come under your care.

December 2nd, 1912.

Effects of Cold Air on Blood-pressures of Children.—Hoobler found that the blood-pressure in children having tuberculosis is persistently low, as has been found to be the case in adults. When a patient is transferred to the open air there is a gradual increase of blood-pressure within one or two hours. If the patient is kept constantly in the open air the pressure is raised to well within the normal limits and sustained at that point as long as the patient remains in the open air. The more advanced the case, the lower the pressure indoors and the higher the rise when put in the open air. After several days in the open air the blood-pressure does not fall as much when placed in ward as it did previous to outdoor treatment.—*Journ. A.M.A.*, vol. lix, No. 20.

THREE LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic; Chairman of the County of London Branch of the British Red Cross Society.

LECTURE III.

RECENT HISTORY, A.D. 1876-1898.

IN a private letter, written some months after the battle of Waterloo by the great Duke of Wellington to his young friend Lady Shelley, he says: "I hope to God that I have fought my last battle. It is a bad thing to be always fighting. While in the thick of it I am too much occupied to feel anything, but it is wretched just after. It is quite impossible to think of glory. Both mind and feelings are exhausted. I am wretched even at the moment of victory, and I always say that, next to a battle lost, the greatest misery is a battle gained. Not only do you lose those dear friends with whom you have been living; but you are forced to leave the wounded behind you. To be sure, one tried to do the best for them, but how little that is! At such moments every feeling in your breast is deadened. I am now just beginning to regain my natural spirits, but I never wish for any more fighting."

These sentiments of the Iron Duke must be shared by every thoughtful man who has seen anything of war, but it is not until one's first campaign that one recognises the immense amount of misery which falls to the lot, not only of the soldier, who may look upon his wounds philosophically, as being all in the day's work, but also of the unfortunate aged folk, women and children in the neighbourhood of the field, who never wished for war, who are driven helplessly away, and who are lucky if they do not eventually find their homestead in ashes when peace is signed.

CARLIST WAR IN 1874.

During the last Carlist War in Spain Sir John Furley found many wounded who had been left for two days without even water or food after the three days' battle of Estella. The Republicans, who on that occasion were defeated, had been so confident of finding all necessaries in the towns that no forethought had been displayed, and the townspeople, on their return would give no help, because their sympathies were all with the Carlists. It was even proposed by some fanatics to burn down a house containing thirty-five wounded Republicans, but fortunately more humane counsels prevailed.

Furley and his servant eventually bought *with gold* an earthenware pitcher, which they continually filled and refilled at a stream till they had assuaged the thirst of 200 wounded in the neighbouring village. When this was done they bought bread and wine in Estella and conveyed them to the village.

The advantages of the Geneva Convention had not penetrated to Spain at this date.

TURKO-SERVIAN WAR OF 1876.

I now come to certain personal experiences which have left a permanent stamp upon my memory.

In the first week of August, 1876, three of us, friends and contemporaries, woke up one morning to find that we had emerged from the chrysalis stage of medical students to the relative freedom of legally qualified surgeons.

Our first duty, we felt, was to visit our chief, Sir William MacCormac, and tell him our interesting news. We had often enjoyed his hospitality and had heard fascinating stories from him of the Anglo-American ambulance in the Franco-Prussian war. When he asked us how we proposed to enjoy our newly earned holiday, we told him that we saw by the newspapers that a war had just begun in Eastern Europe and that we longed to visit the scene of action. By a curious coincidence he had that morning received by post a letter from a benevolent lady, Miss Johnston, asking him if he could find a young surgeon to help the Servians at the front, and offering to pay all travelling expenses.

The spin of a coin decided that your present lecturer was to be the young surgeon, and my two friends went home to obtain the necessary funds to accompany me. During the day we co-opted two other surgeons who had had some war experience at Sedan, and on our journey across Europe we

* Gresham Lectures.

met a medical student of our acquaintance who gladly attached himself to us as a dresser. In this rather haphazard way an ambulance party of five surgeons and one dresser reached Belgrade on August 11th.

To our consternation we then learned that Servian wounded had never in previous history been tended on a battle-field, and when we asked permission to go to the front we had to prove that we were not Turkish spies. Eventually we were given an honorary grade as volunteers in the Servian Army, and were made to don a military uniform. The authorities created further delay by refusing to allow us to leave the capital because we had no swords. No new swords could be obtained for love or money, even by combatants, so we bought six rusty old swords at a second-hand shop, and employed an itinerant knife-grinder to scrub them clean. After our official inspection those swords were packed up in a bundle with our white umbrellas and were never worn again.

My cousin, Dr. Humphry Sandwith, of Kars, was then in Belgrade, and as he was well known to the Servians he was able to inform them about the Geneva Convention, but there were no Red Cross brassards in the country, and we had to start without them.

We jolted in horse waggons along dusty roads till we found ourselves at the front at Alexinatz, eleven days after leaving London. There were further delays due to the staff officers, who did not understand our ambition to get near the fighting line, but on August 20th the Turks drove back the Servian outposts, and before we had had time to absorb the view before us of smoking and blazing villages in the valley below we found ourselves under fire and involved in a panic-stricken stampede.

When the rout was over we formed a dressing station, and then persuaded the general to give us the school-house of the town as a hospital. Fortunately we had with us a plentiful supply of chloroform, for our Servian colleagues had none. We improvised splints, and lined them with hay, for cotton-wool, tow, lint and charpie were very scarce.

Some of us went out every day to the fighting line, and we were gradually imitated by some of the Servian army doctors, who were usually, however, more interested in clerical duties than in surgical work. Russian doctors of both sexes arrived, too, as volunteers, and worked near us with great gallantry and usefulness. We became accustomed to the Servian infantry falling back in most of the engagements, and to finding ourselves unpleasantly within range of the Turkish cannon.

To retreat calmly from a dangerous position with a large body of badly wounded men is no easy matter, and an added difficulty with us was that we had no command of the language, and very little moral control over the terrified and untrained stretcher-bearers who had been allotted to us. The shriek of the shells through the air, the rattle of musketry sounding close at hand, adds, even when there is, perhaps, no actual or immediate danger, to the general alarm and confusion, and it requires no slight firmness to insist on a sufficient body of men remaining at hand to carry the wounded to the ox waggons.

Returning from these excursions, we too often found that there was no food at the hotel, though two well-known war correspondents, Mr. Archibald Forbes and Mr. Fred Villiers, did their best to procure rations for us, and often helped us to tend our patients.

Our hospital, where we spent all our evenings and some of our days, was a house in the main street of Alexinatz, approached by a deep archway, from the sides of which steps went up into the building itself. The street after dusk used to be filled with horse and ox waggons, which had either brought the wounded from the battle-field or were waiting to transport others to the rear.

Beneath the archway the whole space, except one narrow path by which we passed to and fro, was occupied by men lying or leaning, propped against the walls, in every attitude of helplessness and pain. The whole air was filled with their moaning, which among the Servians always had the same peculiarly melancholy note, while above this rose the screams of those in sharper agony.

Within, where the more serious cases were at once taken, the scene was even more sad. Men gasping for breath in their last struggle, others lying helpless and unable to move, stained with the blood that was flowing from them, the dead hardly distinguishable from the dying, except that their moaning was silenced. Happily much could be done for the relief of many. Such dressings as were possible were applied, and the less severely wounded were immediately despatched to the rear,

in order to relieve the pressure for space. Sometimes the task seemed almost hopeless, and as the night wore on an insufficiency of lamps and candles made our work still more difficult.

But, by working hard, each man was in turn attended, all that could be done with the material at hand was completed; the work for the moment was finished.

All this time we had no nurses to help us, but when the other women fled from the town, fortunately one Servian lady, whose home was in Belgrade, determined to remain to nurse her countrymen. Fraulein Spanitz, unassisted by any nurse, proved herself most invaluable to the hospital all through a very trying time. Overworked, unsupported by others of her sex, she stayed at her voluntary post most bravely.

The headquarter staff rapidly became enthusiastic admirers of our methods, and nine days after our arrival at Alexinatz the Russian General in command of the army showed his appreciation of our services by sending to our senior member the Gold Cross for Valour, which had then only been accorded to one Russian and one Servian doctor. Already some 2000 wounded had passed through our hands, though a certain percentage of them were self-mutilated, for cowards found that by shooting off the thumb or finger they escaped serving in the ranks.

The commissariat arrangements were miserable, food was barely procurable and indescribably nasty, and though we had the military power of requisitioning anything we wanted, we often had to buy milk and other articles of diet for our major operation cases in the hospital until they could be transported to the rear.

Meanwhile our friends in London had not been idle. The Order of St. John of Jerusalem in England convened a meeting and persuaded the National Aid Society, who had delayed taking action until they were convinced that the insurrection was not merely local, to vote £10,000 for the sick and wounded Turks and Servians from the balance remaining in their hands after the Franco-Prussian war.

On September 1st this good news was brought to us by Colonel Loyd Lindsay, Sir William MacCormac and others, who had been sent from England formally to take us over as Red Cross surgeons, to furnish us with an ambulance waggon, stores and money, and then to provide a similar ambulance service for the Turks.

While getting together a hasty breakfast for the new comers, we were ordered out to the field, and, after a hard day's work of operations in the ambulance tents (500 wounded), we found at nightfall that the road to Alexinatz had been cut by the enemy, and we were obliged to make a long *détour* with our wounded through roads and villages which were one confused jumble of refugees, children, oxen and poultry. We lived on maize plucked in the fields, did not sit down to a meal for forty-eight hours, and never removed our clothes for a week.

This battle of Alexinatz was the decisive engagement of the war. It lasted for nearly twelve hours, and resulted in a complete success for the Turks. Talks of an armistice followed, and we were ordered back to Belgrade, where we worked in a well-appointed English hospital, which had been opened with deaconesses from the Tottenham Hospital as nurses.

Some of us were at the front again when the Servians unsuccessfully attacked Saitchar, and we spent some uncomfortable days and nights in the rain in charge of an ambulance waggon full of dangerously wounded men. The mountain paths were full of boulders, the pole of the waggon broke more than once, and the horses had to be replaced by a team of eight oxen. On this occasion we had only rum and sticks of chocolate for our patients and ourselves.

We heard at this time by chance of eighty-nine wounded men who had been sent to an out-of-the-way village (Kreevi-veer) and had apparently been forgotten. They were found lying on straw, with no doctor, nurse, surgical dressings, food, nor even water. One was found to be dying of gangrene, but the others were all saved, though another gangrenous case had to have his arm amputated immediately on the arrival of the English surgeons. An incident such as this well portrays the horrors to which the wounded may be subjected when no proper organisation is provided.

Russo-Turkish War, 1877–8.

In April, 1877, war was declared between Russia and Turkey, and during the campaign £30,000 were expended by the National Aid Society on surgeons, stores and steamers in the Black Sea.

Many English surgeons were also employed on the Turkish side by the Red Crescent Society, others were in the pay of the Turkish army, but the greatest number were despatched from here by the Stafford House Committee. The political atmosphere of Eastern Europe had infected England.

Some enthusiasts maintained that oppressed Christians must be aided because they were fighting for their legitimate freedom; others held that our old allies, the Turks, should be assisted, for reliable accounts reported that their armies in Europe and Asia were entirely without adequate medical arrangements.

The Stafford House Committee was formed for the relief of sick and wounded Turkish soldiers, and during two years expended £43,750 of subscribed money. They provided three commissioners, fifty-three surgeons, several dressers and assistants, with hospitals, railway ambulances and soup kitchens, and treated in all 71,274 cases of sick and wounded.

Three of us who had worked together in Servia were among the earliest chosen to proceed to the front, and on August 1st, 1877, we met with our first batch of wounded patients on the railway at Tirnova. There were about 500 of them who had not had their wounds touched since the battle two days earlier. Other trains reached us crammed with fugitive women and children, many of whom had been wounded by sword, knife, bayonet, revolver or carbine. One of our party had to spend part of one night, with a lantern in one hand and a loaded revolver in the other, guarding a van full of wounded Bulgarian peasants whom some Turkish irregulars wished to massacre in revenge for the atrocities committed by the Bulgarians upon Turkish women and children. So far as we could obtain bread, we fed the helpless, and the mothers fought like demons for the loaves.

We then returned to Adrianople, partly because we heard harassing accounts of the wounded there, and partly because the railway lines and bridges had been hopelessly destroyed, and it was necessary for us to get an outfit of horses and waggons for ourselves and our stores to proceed to the front.

We found 2000 wounded in the city, besides more than 10,000 fugitive women and children, and we took temporary possession of a two-storeyed barn about a mile outside the town, in which 200 soldiers were lying on the bare ground, whose wounds had only been dressed once (by us at Tirnova) during the nine days which had elapsed since the battle. The sanitary arrangements were very faulty, but fortunately there were plenty of windows; there was no clean linen, and it took us several days of very hard work to overcome by disinfectants the inodorous condition of the wounds.

During those days we went about in a crouching position because the constant stooping to our patients on the ground fatigued us enormously. But we were rewarded by the gratitude of our patients, who used to cover with kisses the hand which removed the bullet. Several said to us: "The Great God must have sent you here; our own doctors never take this trouble with us, they only ask us where we are hurt and then pass on." We procured linen and straw mattresses and pillows and it was wonderful that only six men died, three of the deaths being from tetanus.

One morning we found, to our horror, that our patients had been brought no food from the city since the previous day at noon. We, of course, complained very energetically, so that the two missing meals were brought on the following day and the uncomplaining patients swallowed them as extras.

There was still a great deal of bad feeling among the Mussulmans and Christians, and some twenty to thirty Bulgarians were hanged every morning to lamp-posts in the principal streets.

The arrival of a fresh batch of surgeons from England freed us for service at the front and we obtained permission to transfer our wounded to a wing of the large barracks adjoining the palace, and leave was eventually obtained to use the Sultan's own bath for the patients of this new hospital, which remained fully occupied until the Turks evacuated the city in the following January.

On August 21st we reached the Shipka Pass of the Balkans with saddle and pack horses, country carts and ox waggons, and for eight days we were busily employed in operating on picked cases from some 10,000 wounded who were sacrificed in vain attempts to storm the Russian position.

Until we had time to put up a Red Cross marquee we suffered from the scorching sun which burnt our bare arms. Absolutely no preparations had been made for the wounded, and

though there were several Greek and Polish doctors attached to the army not one of them possessed surgical instruments, nor could they use those which we gladly lent them. We had annexed in Adrianople a Bulgarian doctor, who was useful in interpreting for us and also in administering chloroform during operations. Upon one occasion when he was thus acting for me he was arrested, in spite of our protests, and marched back to Adrianople, where he was hanged in the street as a spy, with a written confession of his guilt pinned to his shirt front.

Directly there was a lull in the fighting at Shipka we visited Kesanlik, a town in our immediate rear, surrounded by lovely rose gardens, the centre of the trade in attar of roses. The town had been partly burnt and was mostly in ruins, having been subjected in turn to the ravages of the Bulgarians, Russians, Cossacks, Circassians, Turks, and Bashi-Bazouks, but many houses in the Bulgarian quarter were intact and were utilised as hospitals. We were able to supply these hospitals with stores and tobacco, and were invited to operate on several of the patients, many of whom were attacked by hospital gangrene.

At the end of August there were more than 4000 wounded in this little town, some lying helpless in the streets, others imploring assistance from the windows of houses. Typhus fever broke out in the crowded hospitals, and we counted a dozen Bulgarian corpses on the road to Kesanlik, which we never succeeded in getting buried for a month, until Raouf Pasha followed Suleiman Pasha as the general officer in command.

We were fortunately able to give this unhealthy town and its over-filled cemeteries a wide berth when a transport service of horse and ox waggons was organised to take the wounded direct from Shipka to Philippopolis. More than 1800, including some of our amputation cases, were thus evacuated safely to the rear by the energetic Mr. George Stoker. He and his assistants fed the patients, and, when needful, changed the surgical dressings *en route*, and handed the poor fellows over on arrival to English surgeons in charge of the Philippopolis hospitals.

In a final attempt to storm the Shipka Pass 3000 volunteers made a desperate attack upon it, and no less than 1000 of them were brought back to us wounded, some of them by means of a transport service of mule cacolets and hand stretchers, which I had been able to organise for the mountain fighting.

In October we were joined by some English officers who were distributing food and money for the Turkish Compassionate Fund to refugees, some of whom had escaped with sliced wounds at the back of the head from clumsy attempts to behead them.

We were taken by some survivors to an undefended pass in the Balkans, where we found lying in the snow the bodies of 122 Turkish women and children, who had been decoyed there and beheaded and otherwise maltreated by the Bulgarians.

The capitulation of Plevna and the onward progress of the Russian troops through the Balkan passes caused the scene of military operations to be somewhat shifted, so early in January, 1878, we received orders to join the headquarters of Valentine Baker Pasha in the neighbourhood of Sofia. As our small ambulance again travelled towards the front we had ample opportunities of seeing how badly the wounded Turks fared while being transported to the rear. The authorities gave them a sufficiency of hard biscuits, which can only be eaten by a healthy man after they have been soaked in water. The wounded had no other food, and though they were given a little money this was quite useless to them, for, except in the towns, there was no food to buy, and no one to procure it for the sick if it had existed.

The soup kitchens, established by the Stafford House Committee and other charitable societies, were therefore a great boon to those of the sick and wounded who were not fortunate enough to be transported by the ambulance services of such organisations.

When the snow came the ox waggons were converted into rude sleighs by substituting two flat logs of wood for the wheels. In these the wounded glided along very easily and silently.

Continuing our advance, we found that the troops and the population of Ichtiman had been hurriedly evacuated to the rear, and the town was actually in flames before we left it.

The chief Bulgarian inhabitant of Tatar-Bazardjik, who was leaving with his family and servants, handed over to us unconditionally his house, horses, carriages, stores, and poultry, in the hopes that we might be able to save his home from being

burnt. This we were able to prevent, for when we retreated from the town we left the house full of Bulgarian women, who had demanded our protection from irregular soldiers; our Red Crescent flag was flying, and we fixed a written notice to the gate, begging any Englishman who entered with the victorious Russians to protect the house and the women. When the war was over we had the satisfaction of learning that some English journalistic friends on the Russian side had arrived soon after our departure, and had gladly protected the house by occupying it.

When news reached us of the retreat of Baker Pasha's army on Otloukeui, and of the complete loss of medical stores and of most of the doctors, who had been taken prisoners, we were requested by the general in command to proceed to that town. We often suffered from the absence of an intelligence department attached to the Turkish head-quarters, and as we were not placed under any staff officer, we had usually to find out for ourselves where the ambulance would probably be of most value. Upon this occasion we pushed on to the front under orders, only to find that the whole army was in retreat, and that it was necessary for us to leave the town an hour after we had reached it with our horses almost dead beat.

A previous thaw, of two days' duration, had been succeeded by a sharp frost, and the roads were so slippery that the horses were only able to advance very slowly. About one hour from the town the horses of one of our store waggons proved unable to scramble up the thick sheet of ice which coated the whole surface of one of the hills; we left it in charge of a soldier, and continued our journey until the wheel of another waggon came off, and we were obliged to relinquish it also. Soon afterwards the third of our four waggons became fixed in a stream, and the tired horses were unable to drag it out and up a steep hill. About one hour after midnight I camped till morning in the snow, while my friend went off to the nearest village to try and get ox-waggons. I was fortunately able to make a large fire and warm some of the sick and stragglers who reached me during the night; many of these were Arabs, suffering from frost-bitten hands and feet. That night eight soldiers and dozens of horses dropped for the last time near my fire, and died of cold and fatigue. In that month of January the cold was so intense that eighteen of Baker Pasha's sentries were frozen to death in one night, compelling the order that they should be changed every quarter of an hour. In the morning I succeeded in retreating on Tatar-Bazardjik with our remaining stores, but my friend, the dragoman, and some of our servants were all taken prisoners by the enemy, who were in hot pursuit. On this occasion, as on some others, the Russians refused to recognise the sanctity of the Geneva Convention.

I found Bazardjik in a very excited condition: incendiaries were diligently at work, while roving Bashi-Bazouks were looting many of the houses. The telegraph and railway had both ceased to work, the roads were swarming with refugees, and we were ordered to retreat on Philippopolis. On our journey, while crossing a rickety and very slippery bridge, in the early morning light, a cannon fell into the river, closely followed by one of our store waggons; we were only enabled with difficulty to save the horses and stores. We handed over all our remaining heavy stores to the hospital at Philippopolis, and as more than 100 Krupp guns and all wheeled vehicles had to be abandoned, we procured pack saddles, on which our few necessaries could be conveyed.

The road from Philippopolis to Adrianople had been captured by the enemy, and we had to accompany Baker Pasha on a forced retreat across the Rhodope Mountains, by such paths and sheep-tracks as we could find. The hillsides, covered with deep snow, except where the many zigzag paths had their surface beaten into glassy ice, seemed an almost insuperable obstacle to tired men and horses. On every side, upon the ice and in the snow, struggling and fallen horses, soldiers and wounded mixed in frightful confusion, with women and children flying from their burnt villages, all toiled wearily upwards. During the ascent of one extra steep hill, the explosion of an ammunition box, which had fallen from a mule, added not a little to the general confusion. In the plain behind us could be seen the Cossacks advancing to the foot of the hill, and as from there they fired on the struggling masses, soldiers with pack-horses cut loose their baggage, and so hurriedly escaped. Our servants exerted themselves so well that they escaped with the loss of only one box, which unfortunately contained most of our tinned food.

After six days' travelling, without much sleep and with almost no food for ourselves and horses, we reached a small town near the Ægean sea, where we were hospitably entertained by the Greek Bishop and other inhabitants. We then found that 380 wounded men and 120 sick had succeeded in crossing the mountains with us, though we had been unable to do much for them on the march. This was chiefly due to the narrow paths being so blocked with troops that it was impossible for us to keep together or for two men to ride abreast.

On reaching the sea coast we embarked our patients on a steamer which was already crammed with nearly 4000 troops. Nothing was provided for the wounded but biscuit, and it was with great difficulty that we succeeded in obtaining a very inadequate supply of drinking water for them. Some, who were delirious, drank sea-water during our absence and died. The stench of the wounded in the saloon and cabins of this overcrowded ship, many of them still lying in their blood-stained uniforms, was terrible.

Fortunately there came to our rescue Mr. Young, the Chief Commissioner of the National Aid Society, who gave us soup, bread, underclothing and bandages. On reaching Gallipoli we were able to buy oranges and tobacco, for which our patients were deeply grateful, and shortly afterwards they were removed to various hospitals in Constantinople.

The Turkish capital in February was crowded with refugees, and the two large mosques of San Sofia and Sultan Achmet presented an extraordinary sight. The floors of the mosques were peopled by family groups and the matting and bedding which they had succeeded in preserving during their flight; they were fed with bread by officials of the Turkish Compassionate Fund, controlled by Mr. Burdett Coutts. These refugees gave us plenty of work, for smallpox broke out among them, and, until vaccine could be procured from Europe, there were daily many fresh cases among the children.

We visited at this time an almost ironical contrast to the miseries around us. A new barrack hospital, designed by Baron Mundy, had just been opened in Stamboul, and was furnished with clocks, barometers, thermometers and calendars, both in the European and Turkish styles, while every patient had a handsome walnut table near his bed, and the general equipment was marvellously complete. The building was erected by the Red Crescent Society, Germany and Austria fitted it up, the Queen of Saxony supplied the nurses, and an English surgeon was placed in command of the hundred beds. This hospital was purposely placed in the quadrangle of the great Turkish military medical school, in order to familiarise the students with modern surgical appliances and treatment, and regular instruction was given for some months to a large class of medical students. In this extravagantly equipped hospital we were delighted to find some of our former patients.

Time prevents my telling you other notable incidents of the campaign, such as Mr. C. Ryan's single-handed exploits during the siege of Plevna, or the gallant conduct of Dr. E. W. F. Stiven and Mr. W. H. Beresford, who had to remove eighty helpless Turks from the Rustchuk Hospital, which was unfortunately shelled by the Russians on two different days, in spite of the presence of two large Red Crescent flags which were flying at the time; fourteen shells struck the hospital buildings, six patients were struck by fragments of shells, all the servants and other officers of the hospital fled in panic, and there were three feet of snow on the ground at the time.

I must, however, just mention the heroism of Dr. J. Denniston, who, with two of his British colleagues dead and all the three others lying dangerously ill from malignant typhus, remained at his solitary post at Erzeroum in charge of two hospitals containing 500 wounded, and devoted his nights to nursing his delirious assistants.

I have been tempted to dwell at some length upon our Turkish experiences because they are of some interest to-day when we are confronted with another Balkan crisis.

If there is to be another war between Turkey and her Christian neighbours there will again be similar horrors, of which I have given you some slight indication. But the horrors will chiefly be felt by the civilian population.

More than a hundred English doctors were doing Red Cross work on the Turkish side, and of these one third suffered from dangerous illnesses, such as typhus or typhoid fever, 10 per cent. of them died, and a considerable number suffered great hardships by being taken prisoner.

Zulu War and Boer Rebellion, 1879–81.

The War Office invited some of us who had been in Turkey to serve as civil surgeons in Zululand in 1879, but I was so afraid of becoming a rolling-stone that I declined the tempting offer.

The campaign was relatively a small one and the Red Cross aid consisted chiefly in supplying extra medical comforts, hospital equipment and literature, on all of which £3227 were expended.

The operations in Zululand will not be forgotten so long as the annihilation of our troops at Isandula and the heroic defence of Rorke's Drift remain as historical events.

Campaign in Egypt and the Soudan, 1884–5.

Lord Wolseley's campaign to try and relieve General Gordon was the first occasion on which the British National Aid Society to the Sick and Wounded in War was called upon to render help on a large scale to our own troops. Elaborate provision had been made by the War Office, but emergencies arose in the stress of the campaign. Surgeons and nurses, working harmoniously with the officers of the Army Medical Department, dispensed medical comforts with a liberal hand, both on the Nile and on the Red Sea coast. A stern-wheel steamer, acquired by the Society, was very useful on the river in evacuating the wounded and in towing Nile boats full of sick on their return towards Cairo.

Including money collected by the Princess of Wales (Queen Alexandra) and her friends, £34,000 were altogether expended upon surgeons, nurses and material.

One of the features of this war was the great distance that officers and men had to be carried in stretchers and on camel cacolets. For instance, two officers of my acquaintance, both still happily alive and serving their country, had the misfortune to have to submit to amputation of the thigh at Metemmeh after the battle of Gubat. They were safely carried in stretchers for 175 miles across the desert to Korti on the Nile, together with seventy-three other sick and wounded men. The journey was rendered more difficult by the temperature, which was 112° in the shade in February in the Sudanese desert, and the early part of the transport was perilous because a fanatical enemy was harassing the enforced retreat.

Turko-Greek War, 1897–8.

Five English surgeons were dispatched to Greece by the 'Daily Chronicle' National fund for Greek wounded, and it was in this relatively small war that the Röntgen rays were first used in a campaign for the detection of hidden bullets. Temporary hospitals were established in a villa at Phalerum, lent by the Queen of Greece, and in a large one-storeyed building at Chalcis. Both had been occupied by refugees, and required prolonged disinfection to get them rid of vermin. In Chalcis 15,000 refugees were quartered on a population of 9000, and typhoid fever and smallpox were present among them.

The beds consisted of planks on trestles, and the mattresses, in the absence of hay and straw, were stuffed with cotton-waste.

A generous surgical equipment had been provided in London, and the patients were nursed by English sisters, aided by Greek orderlies. The patients had never seen an X-ray apparatus before, for this was the first which had reached Greece, and their superstition sometimes interfered with the skiagrams when the soldiers repeatedly crossed themselves to keep off the evil eye!

The wounded reached the hospital on foot or on horses, for no ambulance waggons could be used in the mountain passes. Many of the patients on arrival had not had their wounds dressed since the action thirty-six to forty-eight hours earlier, but most of them did well. In fact, Mr. Abbott, the chief surgeon, laid down as one of his axioms: "The less wounds are tampered with before satisfactory surroundings are reached the better."

Our Red Cross Society devoted itself chiefly to expending £3600 on medical comforts, and in establishing soup-kitchens for the relief of starving refugees from Thessaly.

In deference to the expressed desire of some of the Gresham audience, I propose to devote my next series of lectures at the end of January to a further consideration of this subject, contrasting, in the wars of the present century, the medical organisation of civilised campaigns with the needless suffering entailed by the absence of forethought among semi-barbarous races.

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THE MODERN TREATMENT OF PHTHISIS.*

By H. WILLOUGHBY GARDNER, M.D., M.R.C.P.,
Senior Physician, Salop Infirmary.

Gentlemen,—The treatment of pulmonary tuberculosis is a very big subject. I can only speak about a small part of it to-night. I cannot speak of all those treatments which we already know so well, and so thoroughly and rightly believe in: cod-liver oil and creasote; good food, warm woollen clothing, healthy dwellings and healthy modes of life; wintering in Egypt, or in the high Alps; long sea voyages; the climates of South Africa, Australia or Colorado; and best treatment of all, life spent entirely in the sunshine and the open air. To-night I want to discuss the more special advances of the last few years—our efforts, if I may so speak of them, to come to closer grips with the enemy.

Knowledge came in from many quarters. The various problems of phagocytosis and immunity had been studied; toxins, the products of harmful bacteria, had been discovered, and the antitoxins produced by the body in its efforts to resist them; later on, other anti-bacterial substances came to be known—agglutinins, bacteriolysins and opsonins. There is no doubt that the work of Wright with his opsonins shed great light on the problems of infection and immunity, and his discovery of vaccines greatly added to our therapeutic resources and to the hopefulness of our work.

Tuberculin continued to be studied by Koch and his pupils, and our knowledge as regards its powers and its dangers and the best methods of employing it steadily increased.

* A paper read as Presidential Address before the Shropshire and Mid-Wales Branch of the British Medical Association.

It had long been noticed that phthisical patients varied much in their condition from time to time, especially those who were feverish. Their charts showed long waves of raised temperatures, corresponding to a bad clinical period, and long waves of lower temperatures corresponding to an improvement in the patient's general condition. It was found that the good times were times of increased immunity—times when the blood had become loaded with antitoxins and other anti-bodies—and that the bad times were the times when the immunity was lessened, the antitoxins were diminished, and the toxins in the blood got the upper hand in the struggle. As long as the patient's vitality was sufficient, these good times and bad times alternated like the swing of a pendulum, the good times being due to the gradual reaction of the body against the excessive toxins of the bad times.

Again, it was found that many patients in a fairly quiescent state were liable to feverish attacks closely resembling influenza; that these attacks would often leave them weaker than before, and often with an increase of the physical signs in the lungs.

Some few patients on the contrary would be the better for these attacks, and would even date their improvement from one of them. Why was this?

Gradually it was learnt that the attacks were not influenza or ordinary colds; were not due to infection from without; but closely followed on any over-exertion and fatigue; followed less often perhaps some exposure to cold and damp. Gradually it was perceived that they were due to poisoning with the patient's own tuberculous products; auto-intoxication due to an overdose of his own toxins. In the cases where he was left worse after them it was because the dose of poisons lowered his vitality and lessened his resisting powers and so his disease increased and spread. If, on the other hand, the patient improved after them, it was because he was able to meet the extra dose of tuberculous toxins by a sufficient and more than sufficient production of antitoxins and other anti-bodies; so the disease did not spread, and when the febrile attack was at an end he had a supply of anti-bodies left over; he had gained increased immunity, and so for a time he was superior to his disease—it had lost its power to harm him. During that time of immunity he was able to regain health and strength, to rise to a higher level.

Soon it was found that if we let our patients go about as usual during these occasional febrile attacks, they were almost sure to be the worse for them, but if we kept them absolutely at rest in bed for a few days, until the attack was well over, the patient would generally be the better for it.

So the thought came, Was it possible to regulate these attacks of auto-intoxication with the production of anti-bodies resulting therefrom so that they might be used to raise the patient to ever higher and higher levels of health, and enable him to conquer his disease?

The next thought was, Could we, instead of stimulating the production of anti-bodies by auto-intoxication, produce the same effect by injecting tuberculin in carefully measured and regulated doses?

All our best modern treatment has come from the attempt to answer these two questions.

Both methods are now used, namely:

(1) The method of producing efficient auto-inoculations and regulating the response thereto by graduated exercise and rest.

(2) The method of provoking our immunising responses by carefully injecting tuberculin in adequate and gradually increasing doses.

There are strong advocates of each system.

Treatment by graduated rest and exercise has been carried out with striking success by Dr. Paterson at Frimley Sanatorium, and in one form or another is now used, I believe, at most other public sanatoria.

Tuberculin treatment has been used widely in Germany for many years, and during the last two or three years has been largely used in England. It is now employed, I believe, in most sanatoria, and in several large out-patient dispensaries, and it is likely to be widely used under the new scheme of sanatorium benefits.

It may be well to compare the relative advantages and disadvantages of the two methods.

The first method, that of regulating auto-inoculations by graduated rest and exercise, is certainly efficacious, and is especially useful for those who will have to earn their own living by manual labour after they leave the sanatorium. They leave at the end of their treatment no longer delicate invalids, but healthy and strong, with firm muscles and

strong hearts, cheerful and confident. They are able to earn their own living, and are determined to do so. They have learnt not only the value of open-air life and healthy conditions of life, but also the value of exercise and of rest. They have learnt how to regulate their own lives so as to keep well. They know there is no special mystery about it. They are independent of doctors and of drugs, and they are not only more resistant to phthisis, but also to all other diseases.

The disadvantages are that it needs the most constant supervision and the strictest discipline, and it needs the enthusiastic co-operation of the patients themselves to ensure success. It is almost impossible to carry out in ordinary private practice, or even in hospital practice, owing to the extreme difficulty in duly regulating the amount of rest and exercise, *i. e.* in regulating the doses of auto-inoculation, and the immunising responses of the body thereto.

The advantages of the tuberculin treatment are that the exact dose needed is given by the doctor himself at exact intervals, so as to produce an exact effect.

The disadvantages are that the patient is not himself helping in his cure; he is not able to go on with it unaided in after life; he is not turned out hard, and muscular, and fit for his daily work, but is liable to drift into a state of invalidism, and to lose his energy and vigour. He may thereby be rendered immune to tuberculin, but he is not made strong and fit to cope with the battle of life, and able to resist the innumerable hostile influences which will surround him.

In most cases it is best to combine the two methods, as is done by Dr. Pearce so successfully at our own sanatorium. The disadvantages of this combined method are that it is not so scientific; its problems are not so simple; it is not so easy to tell whether a rise of temperature is due to an overdose of tuberculin or to some over-exertion producing auto-inoculation; whether improvement is due to the tuberculin we are using, or to the effect of the healthy outdoor exercise upon the patient's general health. So it is more difficult to regulate our dosage of tuberculin, and to regulate the amount of exercise taken; the possibilities of error are increased.

The method is a compromise; but, like many compromises, it works well. In practice it is best to think of our treatment in terms of tuberculin, and to use the rest and the graduated exercise merely as a help. Experience soon teaches us how best to combine the two, and to put our finger on the spot if anything goes wrong. Practically, as we shall see, we are generally able to increase our patient's tolerance so greatly by the use of graduated doses of tuberculin, that any extra dose of his own autogenous tuberculin produced by some little over-exertion falls practically within the limits of that tolerance, and is felt little if at all.

The great advantage of this combined method is that we are able steadily to increase our patient's tolerance by our tuberculin, and so to give him immunity against his disease, and by our exercises we are increasing at the same time his muscular strength and bodily and mental vigour; so we shall turn him out in every way a healthier, a stronger, and a better man. The tuberculin treatment we can continue after he leaves the sanatorium. The rest and exercises he will have learnt to manage for himself. Anyone visiting our sanatorium, and seeing the patients cheerfully working there, and seeing their vigorous, healthy appearance, will, I feel sure, be satisfied with the results of this combined treatment.

To get good results from either of these methods, or from both, it is necessary that everything should be done to increase the patient's general vigour and health. For the success of the treatment is due to a vital response on the patient's side, and he must be able so to respond. Therefore we give him everything we can to invigorate him—life in the open air, good and plentiful food, warm clothing, cheerful occupation, and cheerful surroundings. If we can give him warm sunshine, a dry soil and dry bracing air to live in so much the better. At the same time we must carefully remove everything harmful; must especially see that he has no bad teeth causing septic poisoning; must cure any indigestion, and relieve any useless cough —which is in itself a potent cause of irregular auto-intoxication; and must try to remove all anxieties and worries.

We will now discuss these two great methods of treatment.

We will take first the treatment by graduated rest and exercise, because it is far the simpler, and when we understand it thoroughly, it will be easier for us to master the treatment by tuberculin.

Dr. Paterson points out that it is not the actual amount of disease in the lungs that is of real importance, but the balance between the amount and virulence of the toxins produced by the bacilli and the resisting powers of the body. This balance is shown by—

(1) The temperature.

(2) The amount and appearance of the sputum.

(3) The patient's appetite, and feeling of well-being or the reverse.

(4) His gain or loss in weight.

If he suffers from excessive auto-inoculation, *i. e.* if the dose of toxins is too great for his resisting powers he will get—

(1) A rise of temperature.

(2) He will feel ill, and lose his appetite; have headache, and pains in his eyes and limbs.

(3) There will be an increase in the amount of sputum.

(4) And he will lose weight.

The aim of the treatment is to raise the patient's resisting powers, so that he can perform the hardest work without it producing constitutional disturbance, and at the same time to bring him up to his greatest known weight.

The way in which this is done is:

First, any excessive auto-inoculation must be controlled by absolute rest; so as to limit as far as possible the amount of toxins thrown into the circulation, and thus enable the resisting powers of the blood to deal with, and overcome, those already absorbed. To enable this to be done rapidly and effectively, Dr. Paterson insists that the rest must be absolute; patients must be treated exactly like typhoid fever patients—not even allowed to sit up in bed or to feed themselves. It is wonderful how quickly patients react to this strict treatment; it is by far the most efficient treatment of fever in phthisis yet known. In about three or four days as a rule under this treatment the temperature will have become normal; after two or three days longer of ordinary rest in bed the patients are allowed to get up, and carefully each day to take more and more exercise. It is found then that they are the better and not the worse for their attack of fever. In the victorious struggle against the excessive dose of toxins they have gained increased immunity, and after a few days they are put back to the same grade of work at which they had broken down, and it will be found that they are able to manage it without any bad symptoms.

This absolute rest on any appearance of excessive intoxication is the sheet-anchor for all successful treatment. It is just as important if treatment is by tuberculin as if it is by graduated rest and exercise. It is just as important in hospital practice, and in private practice, as in a sanatorium; and if our private patients can be made to understand its importance, and be induced to carry it out faithfully, we shall get as good results with them as in sanatorium practice. These attacks of excessive auto-intoxication are bound to come; in the old days they marked steps in the downward course of our patients. If made use of in the way Dr. Paterson has taught us they will more often be steps in his upward progress; he will often trace his most marked improvement to one of these bouts of fever.

This control of excessive intoxication is perhaps the most important advance of all in our modern treatment; it is this, the real value of rest, which I want you all thoroughly to appreciate.

Next we come to the graduated exercise. The value of exercise is to increase the amount of toxins which get into the blood from the tuberculous focus, and so to stimulate the body to produce antitoxins and other anti-bodies wherewith to fight the disease.

This exercise is carefully graduated so as never at any time to cause a bigger dose of toxins to get into the blood than the protective mechanisms of the body are able to deal with. If this is done carefully and gradually enough, these protective mechanisms gain ever-increasing power, and are able to deal with bigger and bigger doses, and so the patient's immunity and all the resisting powers of his body are gradually increased, and he becomes more and more able to cope with his disease.

If at any time, as will happen, the dose of toxins set free is too great for his resisting powers, the patient will show the symptoms of auto-intoxication—fever, malaise, etc. Then he must without delay be once again treated by absolute rest until the balance has been restored.

It will be seen that for the success of this treatment by exercise the patient must be afebrile to begin with. If he is febrile he is already getting too much auto-intoxication. He then wants rest,

not exercise, and until the fever has disappeared treatment by graduated exercise cannot be begun. Fever needs rest; the more complete the rest the sooner will the fever disappear. But disappearance of fever does not mean cure of the disease. A patient treated by rest alone—apparently successfully—though he may look well, feel well, have lost all fever, and have gained weight will not have gained tolerance or immunity. When he begins work again he will sooner or later get a dose of auto-toxins greater than he can deal with, his fever will return and he will break down again.

We should try to increase his tolerance by our graduated exercises, until it reaches a point far beyond the necessities of ordinary daily life and work; then he will be able to gain his own living once again.

Dr. Paterson begins his treatment by graduated walking exercise.

(*a*) *Those who have had fever* begin with half a mile a day, and spend the rest of their time resting, thoroughly and faithfully. Then they gradually increase the distance to one, two, four and six miles a day, taking some five weeks to do this.

If any bad symptoms supervene the patient is given an interval of rest and then kept on the same grade until all symptoms have subsided.

(*b*) A patient who has not had fever may begin on two, four, or six miles a day, according to his condition and the amount of his sputum. He is kept a week on each grade in order that his muscles may develop and be equal to that amount of exercise and do it easily.

After the six miles' grade has been completed they pass into the first grade of labour. They now begin to exercise the upper limbs and upper part of the body, and so bring the lungs, the site of disease, more directly into play.

Dr. Paterson has six grades of labour, beginning by carrying small baskets weighing 10 lb. some fifty yards at a time, and ending with five hours a day at the hardest navvy work, with one hour a day indoor work. All the work done is useful and productive, and is, therefore, pleasant work.

Then before going home the patients are set to work for a time at their own occupations, whatever they may be, so as to get the proper muscles accustomed to the work.

Thus they are sent home in grand health, muscularly strong, fit and able to do their own work and once again to earn their own living, having gained immunity against any dose of toxins or other bacterial products which they are likely to receive. They have also learnt how to take care of themselves, to keep in good health; they have learnt the value of fresh air, and good food, and rest and exercise, and how to use these methods for their ultimate restoration to health; they have probably not got rid of all their tuberculous disease, but they have gone a long way towards discovery. If they can and do keep well and strong Nature will eventually effect a cure, since pulmonary tubercle is one of the most curable of diseases, and its tendency is to die out. Meantime our patients have also learnt what precautions are necessary to prevent the spread of infection to their families and fellow-workmen and have got into the habit of using these precautions.

We must next consider the treatment of phthisis by means of tuberculin. The general principles are the same. Beginning with small doses, and gradually and very carefully increasing our doses so as always to keep within the limits of tolerance, we aim at gradually increasing the resisting powers of the body until we have bestowed upon it a high degree of immunity. Immunity to our tuberculins means a corresponding immunity to the patient's own autogenous tuberculin toxins. Our aim is to raise it to such a high degree that any dose of auto-intoxication they may get, even after severe exercise, will be far below the limits of the tolerance they have gained, and so will be unfelt by them—will cause no constitutional symptoms. In this way they will be raised up above the influence of their tuberculous disease, will be practically unaffected by it, and will have every chance of recovering from it.

It was hoped that by the use of some variety of tuberculin we might be able to do far more than this; that we might be able to call out some bactericidal properties (substances), and thereby actually destroy the tubercle bacilli *in situ* and so cure the disease. To a limited extent this is practically accomplished indirectly whatever form of tuberculin we use, but on the whole the result so far has been disappointing. We can do far more to counteract the toxins produced than to destroy the bacilli. Still, if the patients get well, and keep well, the bacilli do tend to die out, and

the disease to become cured, as is so often to be seen in our post-mortem rooms.

A few words as to the various forms of tuberculin. They are very many. The great thing to remember is that the effect of them all is in the long run practically the same. Treatment may be, and often is, successfully carried out with any one of them. It is best at first to chose some one form of tuberculin, pin one's faith to it, and learn thoroughly how to use it. Still, there are certain things we all ought to know.

First of all there is the great division into human and bovine tuberculins. A vast amount of discussion has raged round the question of human and bovine tuberculosis. The question is not yet, I think, finally settled. The probability is that the human and the bovine tubercle bacilli have been originally the same species, and that they have become differentiated by passing for generations through man or cattle; that the bovine bacillus is far less virulent for man than is the human variety; and that the bacterial products of the bovine bacillus are far less toxic for man than are those of the human bacillus.* Therefore, it has been thought by many best to use bovine tuberculins for treatment instead of human tuberculins, at any rate in the early stages of the treatment. That is the system pursued by Dr. Camac Wilkinson and by many of his followers throughout the kingdom, at Dr. Fraser's tuberculin dispensary at Portsmouth, and, I believe, by Dr. Wynn at Birmingham.

There seems little doubt that the reactions produced by bovine tuberculins are of shorter duration, and are as a rule less severe. The period of raised tolerance is therefore shorter, so that it seems very important if using them not to let the intervals between the doses be too long.

These bovine products are distinguished by the letter P. = Perlsucht. Thus we speak of P.T.O., P.T., P.B.E., etc.

The next great division of the tuberculins, and one very important for practical purposes, is into soluble and insoluble products. Of the soluble products, old tuberculin, known by the symbol T., is the type. It is Koch's original tuberculin. It still holds its own and is greatly used for various purposes. Most of all it is used for purposes of diagnosis—for the various tests such as Von Pirquet's and the subcutaneous injection tests. It is essentially a filtered extract of a pure culture of human tubercle bacilli, and contains, therefore, the extra-cellular, soluble products of those bacilli, which are the most toxic.

Of the insoluble products Koch's new tuberculin (known as T.R.) is the type. It is made by grinding up the tubercle bacilli themselves into a fine emulsion after the soluble products—which are so toxic—have been washed away. Koch hoped when he produced it that he had found a tuberculin which would have a definite bactericidal action. In this, however, he was to a great extent disappointed, for the results of treatment by it have proved practically the same as those by the other forms of tuberculin. It has, however, these great advantages. It is a very mild form of tuberculin, less likely to cause toxic reactions, and, being insoluble, its action is slower and more prolonged. It is very useful in the treatment of surgical tuberculosis, which I hope to say a few words about, and also for delicate and highly susceptible patients, with whom we have to proceed with the utmost caution. One disadvantage is that it is expensive.

The next form of tuberculin to speak about is Koch's bacillary emulsion B.E. It contains the *whole* products of the human tubercle bacillus, both the insoluble and the soluble tuberculin. It is therefore exactly like all the vaccines we are accustomed to use. By many people it is thought to be the most efficacious form of any. Dr. Camac Wilkinson, however, and some others tell me they have given it up because they found it occasionally to produce very severe and dangerous reactions. It is hardly fitted, I think, for out-patient practice, except in the case of strong, hardy subjects; and needs watching carefully. It has one great advantage—it is cheap.

If I might advise you, I would say:

(1) Use bovine tuberculins, especially P.T.O., for out-patient practice, where you can be sure of seeing your patients twice a week.

(2) Use T.R. in cases of surgical tuberculosis, and for very delicate and susceptible patients, and also in febrile cases, as it is the safest and gentlest form.

(3) Use B.E. as a routine practice for all strong patients whom you have completely under your observation and control, and even for strong out-patients, and perhaps for the later stages of treatment begun with bovine preparations.

(4) Use T. for diagnostic purposes, and for the later stages of treatment by bovine preparations.

Before discussing the administration of tuberculin there are certain general considerations and terms which we have to consider.

We speak of (1) sensitiveness; (2) tuberculin reaction; (3) immunising response; (4) tolerance; and (5) immunity.

We must try to know clearly what we mean by these terms.

(1) *Sensitiveness:* By this we mean sensitiveness to tuberculin. It is not at all the same thing as susceptibility to infection by tubercle bacilli. That is a thing I cannot discuss to-day, though it is a most interesting subject. This *sensitiveness* is shown by a reaction to tuberculin, when it is applied to skin or mucous membrane or injected into the tissues. Healthy, sound people show no such sensitiveness. In them the injection of tuberculin, even in large quantities, produces no effect whatever. The presence of this sensitiveness shows, therefore, that the patient has tuberculous disease somewhere about him; or it may be, has had; for this sensitiveness may persist even for some years after the tuberculous disease has become completely cured. It is the presence of this sensitiveness which we make use of for our tuberculin tests—Von Pirquet's, Calmette's, and the subcutaneous test.

Sensitiveness varies greatly; it is apt to be the same in the same patient, for long periods, if unaltered by treatment; but it varies enormously in different persons, certainly a hundredfold, possibly even a thousandfold. We can increase it or decrease it by tuberculin treatment. It is in itself a useful thing; is actually a step towards immunity.

People who are already "sensitive" are less liable to fresh infection, or to infection of fresh parts of the body, than those who are not sensitive; or, in other words, those who have already a tuberculous focus are less liable to develop phthisis or general tuberculosis than those who have not; they are to a certain extent protected. This is a matter of clinical knowledge. Years ago it was the custom to remove tuberculous glands, hip joints, etc., by operation, "so as to remove the source of infection." The results were disastrous. Children who had thus been operated upon often developed phthisis, and still more often general tuberculosis. Now, it is extremely rare for children with tuberculous joints or glands, properly treated, to develop phthisis or general tuberculosis. They are protected thereby, though found to be very sensitive to tuberculin. It is found also that men coming from districts like the Steppes of Russia, where tubercle is absent or rare, and who are unprotected by this sensitiveness, are very liable to succumb to phthisis when they come to live in towns or in army barracks; more so than town dwellers, 70 to 90 per cent. of whom are found to possess sensitiveness, as shown by Von Pirquet's test, and therefore to possess some degree of immunity.

Experimentally, too, it has been found that if some animals which have already a tuberculous focus, and others which are free from tuberculous disease of any kind, are injected with virulent tubercle bacilli, the former being sensitive show an immediate reaction, but no further harm follows, while those which are free from tuberculous disease show no reaction, but soon succumb to general tuberculosis.

How, then, does this sensitiveness protect them? Well, it acts like a police force. When tubercle bacilli are introduced at any spot sensitiveness causes an immediate local reaction at that spot to fight the disease locally, and a general constitutional reaction to fight the toxins and any tubercle bacilli introduced into the blood.

This brings us to the *tuberculin reaction* which sensitive beings show to tuberculin. This *reaction* is divided into local, general, and focal.

The *local reaction* consists of inflammatory redness and swelling of the skin at the seat of injection.

The *general reaction* is shown by rise of temperature and other constitutional signs, such as headache, loss of appetite, and general malaise.

By *focal reaction* we mean an inflammatory reaction at the seat of the tuberculous disease itself, easily visible if the disease is on the surface, discoverable by signs and symptoms if the disease is in deeper parts of the body.

These reactions are more or less severe according to the sensitiveness of the animal and the dose of tuberculin. They last for a longer or shorter time according to the dose and according to the power of the animal to respond to it. They correspond

to the negative phase of Sir Almroth Wright, a period during which all the vital powers of the body, and *especially the powers of withstanding tubercle bacilli*, are depressed.

They are followed after a longer or shorter time by what is known as the *immunising response.*

While this immunising response is occurring the patient's blood is full of anti-bacterial substances, so his power of fighting the tubercle bacilli is greatly increased. The local diseased area is probably still flushed with blood, and the blood is now loaded with anti-bodies, harmful or destructive to the tubercle bacilli; phagocytosis is actively going on there, the phagocytes being aided by the increase of opsonins in the blood. The patient's general health is improved for the time, he eats well, sleeps well, feels well. his pulse and temperature are normal and he soon begins to gain weight, and the lesions themselves may often be observed to improve rapidly. This period of the immunising response will last a longer or shorter time. When it is over, a second similar dose of tuberculin will produce similar effects, except that we start each time on a higher plane of general health, owing to the good effects of each previous dose of tuberculin, and so progress is made towards the cure of the disease.

This is what happens if the disease is strictly localised, and the part affected kept absolutely at rest. The best way to treat such local disease is by a small dose of tuberculin once a fortnight. This dose, if found to be suitable, is not increased: the same dose is given again and again. We must, however, secure absolute rest for the joint or other affected part, so that the patient may get no auto-intoxication.

But in phthisis the conditions are quite different. Here the disease is in a very vascular organ, which is also in constant movement. Hence auto-inoculations are constantly occurring; toxic bacterial products are constantly being discharged into the blood in ever-varying quantities. Auto-tuberculin reactions constantly follow, and there are more or less constant attempts at immunising response which may or may not be successful, according to the doses of the toxins, and according to the resisting powers of the patient. Here, as we have seen, we may do good in two ways: first by rest we may diminish the amount of toxins poured into the blood, and secondly by fresh air, good food, etc., we may increase the vital powers of our patient.

But there is something more we can do, and which we must strive to do. We want to develop in our patient *tolerance* for tuberculin, and to gradually increase that tolerance until it has become so great that any dose of auto-tuberculin he can possibly get from the tuberculous disease in his own lungs is not felt by him—causes no bad symptoms. Then his general health and strength will improve and his disease will tend to die out, for the vital powers of his own tissues, aided by the anti-bacterial bodies, the bacteriolysins, agglutinins and opsonins which our tuberculin reactions have called forth in his blood will be able to overcome it. This, as we have seen, can be done by the method of graduated rest and exercise; it can also be done by the help of our tuberculins. We gradually increase our patient's tolerance by giving carefully graduated, ever-increasing doses of tuberculin, and so we give him ever-increasing immunity.

We may say he has acquired sufficient *immunity* when the largest dose of bacterial products which the most violent exercise can throw into his blood can no longer produce constitutional symptoms. This *immunity* does not necessarily mean cure of his tuberculous disease, or even the total absence of tubercle bacilli from his sputum; but as a rule it is found that such a one has no sputum and coughs up no tubercle bacilli, and that his disease is far less in amount and is quiescent and tending to become extinct. If we can keep up this immunity long enough our patient will probably get quite well and keep well, and show only the "scars" of his past disease.

But a patient who still has tubercle bacilli in his sputum may be in quite good health as the result of treatment. He is then analogous to a typhoid carrier; he is a tubercle bacilli carrier, but he is *immune* to his own bacilli. I shall hope to show you such a one at the next meeting of the Clinical and Pathological Section.

After each dose of tuberculin when the immunising response begins, *tolerance* begins to rise; it falls gradually as the immunising response comes to an end; it is, however, capable of being built up almost indefinitely by steadily increasing doses of tuberculin given at proper intervals; and the greater it is, the longer it will last.

We must now consider more definitely how we are to use the special form of our tuberculin which we may have chosen in cases of phthisis.

We have to consider—

(1) What is to be our initial dose.

(2) How much we are to increase the dose each time.

(3) How often we are to give it—*i. e.* what is to be the interval between the doses.

(4) What is to be our maximum dose.

(5) What we are to do when we have reached the maximum dose, and how long we are to continue our tuberculin treatment.

(6) What clinical indications we are to look for to guide us throughout our treatment.

First, however, it seems to me that we want some rough classification of our patients. The old classifications according to the amount and duration of the disease are useless for our purpose. We want one which will help us to estimate—

(1) The amount of toxins which are getting into our patient's system.

(2) His power of successfully reacting against them.

The best classification I know is that given by Dr. Morland and Dr. Riviere in their book.

They divide patients into three great classes:

(1) *The resting febrile* — those who have a raised temperature, *i. e.* symptoms of auto-inoculation, even when at rest.

(2) *The resting afebrile, ambulant febrile*—those who have no fever when kept at rest, but get a raised temperature when they walk about.

(3) *The ambulant afebrile* — those who get no fever, *i. e.* no symptoms of auto-inoculation, even when walking about.

The first class, those who have fever even when lying quietly in bed, I shall say nothing about at present. Their treatment bristles with difficulties.

The second class, those who are afebrile only when resting completely, are particularly well suited for tuberculin treatment; but during its early stages they must be kept strictly in bed—kept in bed until, as a result of the tuberculin treatment, they have gained so much tolerance that any auto-inoculations they may get when they first begin to move about will fall well within the limits of that tolerance and so will not cause fever or constitutional symptoms. This will require much care and judgment, especially when first such patients begin to get up.

In the third class, those who get no fever even when walking about — tuberculin treatment is generally easy and very successful. It is well to gain one's experience by treating at first only such cases.

Next we must consider the measurement of the doses and how they are to be given.

There has been great confusion about this, owing to the many different measurements used. I personally have become accustomed to think in fractions of a milligramme (the old method). It is quite irrational, for a milligramme is a weight for solids, and we are using liquids and want measures, not weights.

I strongly recommend you always to use decimals of a cubic millimetre as your standard of measurement. Remember it is cubic measure, and a cubic millimetre is a thousandth part ($10 \times 10 \times 10$), not a tenth part of a cubic centimetre.

Then one uses the tuberculin in six different dilutions—for convenience—each dilution being one tenth the strength of that immediately above it. The dilutions should be made fresh at least once a fortnight; the pure tuberculin keeps indefinitely, but the dilutions rapidly lose strength, and so trouble may arise whenever a fresh dilution is made—its strength being far greater than it should be in relation to that previously used. It is very easy to make these dilutions. One wants some diluent fluid—$\frac{1}{2}$ per cent. carbolic acid solution, sterilised; for each weaker dilution in the scale $\frac{1}{10}$ of the previous one is added to $\frac{9}{10}$ of the diluent fluid. This is easily measured with our syringe. The accompanying table gives the relative strength of the more common preparations in reference to the old measures by weight. Fortunately all we have to remember now is the first column and the dilution which corresponds to it, which is shown in the third column. It comes right for all the preparations we are accustomed to use.

Next comes the grading of the doses.

We use a syringe, preferably all glass as being easily sterilised, containing 1 c.c. and graded in $\frac{1}{10}$ of a c.c.; each tenth is spoken of as one division (of the syringe). The syringe must be of narrow bore and each division should be subdivided into halves.

Now it is very evident that if we use as our doses $\frac{1}{10}$, $\frac{2}{10}$, $\frac{3}{10}$, etc., we shall be doing wrong; $\frac{10}{10}$ is only a tenth more than $\frac{9}{10}$, but $\frac{2}{10}$ is twice $\frac{1}{10}$

*Table of Dosage for the various Tuberculins.**

Cubic mill metre c.mm.	Cubic centimetre c.cm.	Contained in 1 c.c. of dilution.	Equivalent in older systems of measurement. T., P.T., P.T.O. in milligr's.	T.R. in milligr's.	B.E., P.B.E. in milligr's.	Beranek's T.B.K.		Remarks.
1000	1	Original strength	1000 mg.	10 mg.	5 mg.	—	—	—
100	0·1	I	100 mg.	1 mg.	$\frac{1}{2}$ mg.	T.B.K.$_{.1}$ 1·0	—	This may be considered the ordinary maximum dose.
10	0·01	II	10 mg.	$\frac{1}{10}$ mg.	$\frac{1}{20}$ mg.	T.B.K.$_{.2}$ 1·0	—	When this dose has been reached the interval may be gradually increased.
1	0·001	III	1 mg.	$\frac{1}{100}$ mg.	$\frac{1}{200}$ mg.	T.B.K.$_{.3}$ 1·0	—	—
0·1	0·0001	IV	$\frac{1}{10}$ mg.	$\frac{1}{1000}$ mg.	$\frac{1}{2000}$ mg.	T.B.K.$_{.4}$ 1·0	—	—
0·01	0·00001	V	$\frac{1}{100}$ mg.	$\frac{1}{10000}$ mg.	$\frac{1}{20000}$ mg.	T.B.K.$_{.5}$ 1·0	—	·05 may be considered the average dose in an adult for surgical tuberculosis.
0·001	0·000001	VI	$\frac{1}{1000}$ mg.	$\frac{1}{100000}$ mg.	$\frac{1}{200000}$ mg.	T.B.K.$_{.6}$ 1·0	—	We may take this as our initial dose in all ordinary cases. It is best given as one division ($\frac{1}{10}$) of No. V.

The cubic millimetre is used as the standard of measurement.

*Scheme of Dosage for Tuberculin (graduated so that each dose is the same multiple of the preceding one).**

10·0	10·0	10·0	10·0	10·0	10·0	10·0	10·0	10·0	→Divisions = 1 cubic centimetre.
							8·3	8·6	
			6·3	6·8	7·5	7·9	6·8	7·4	We may consider Table No. X as our ordinary guide in ordinary cases. If patients are very delicate and very sensitive we use Table No. XII or No. XV. If patients are strong and not very sensitive and we begin with very small doses we push rapidly ahead, using Tables No. III, No. IV, or No. V, until a reaction occurs, and then go steadily on with No. X.
		5·6			6·3		6·8	6·3	
	4·6				5·6	6·3	5·6	5·4	
			4·6	4·6		5·0	4·6	4·65	
			4·0		4·2	4·0	3·8	4·0	
3·2		3·2		3·2	3·2	3·2	3·2	3·4	
			2·5			2·5	2·65	2·9	
	2·2			2·2	2·4		2·2	2·5	
		1·8			1·8	2·0	1·8	2·15	
			1·6	1·5		1·6	1·5	1·85	
					1·3	1·3	1·2	1·6	
								1·4	
								1·2	
1·0	1·0	1·0	1·0	1·0	1·0	1·0	1·0	1·0	→Division = $\frac{1}{10}$ cubic centimetre.
Scale No. II	III	IV	V	VI	VII	X	XII	XV	= The number of the series, or "Tables."

N.B.—This table is correct for each of the six dilutions.

* These tables are adapted, by kind permission, from Riviere and Morland's book on tuberculin treatment.

so we are obliged to have tables worked out for us by mathematicians, to show us what the steps in our doses are to be. The size of these steps varies in different cases, but each successive step must be of the same size. The accompanying table shows the various scales we use.

If our patient is strong and not sensitive and has no fever we can go ahead rapidly, using scale Nos. III, IV, or V, until we get a reaction, when we use scale No. X, which we may take as our ordinary guide in average cases. If, however, our patient is very delicate and very sensitive, getting reactions very easily, we must go very carefully and gradually to work (using scale XII or XV). Of course, we shall then get on far more slowly, but we must be content to do so.

We may take ·001 of a cubic millimetre as our ordinary initial dose, best given as one division of No. 5 dilution. In ordinary cases we push rapidly ahead, giving a dose twice a week, and increasing the dose on the basis of table No. 3, *i.e.* giving 1 division, 2·2 divisions, 4·6 divisions, 10 divisions, practically doubling the dose each time, and so on until we get a reaction. When we get a reaction we know that our tuberculin is causing a therapeutic effect. The reaction we look for is the local skin reaction at the site of our injection. This is a far more delicate test—perhaps ten times more delicate—than the general reaction. We must, if we possibly can, avoid getting general reactions at all; they may do harm, at any rate may hinder our progress; they show that we have given too big a dose. The local skin reaction is a sure sign of a focal reaction at the site of the tuberculous lesion itself; some think that they correspond very closely. As soon as we get a local reaction we advance much more cautiously and slowly, using scale No. X as our guide for the increase of our doses, but still allowing the same interval of time between our doses.

If perchance we get a general reaction, *i.e.* rise of temperature, or even any unpleasant constitutional symptoms without a rise of temperature, we must pause in our progress; we must wait until the reaction is completely over, perhaps one, two, or three days longer, and then if the constitutional reaction has been very slight repeat the same dose.

If, however, the general reaction has been at all severe, or if a second similar dose causes a greater reaction than the previous one, we must wait until the reaction is well over, and then give a much smaller dose, say from $\frac{1}{4}$ to $\frac{1}{10}$ of the dose previously given, and again steadily increase it. Probably this time we shall find that we can go ahead all right once again, perhaps to come to a similar check a little later on.

If constitutional symptoms again occur, but are slighter, we must again wait and again repeat the same dose, and so on, until we can give it without its causing a reaction, and then go ahead again on the X basis as before.

If we find that reactions keep occurring as we increase our doses, then we must be content to progress more slowly, using No. XII or even No. XV scale as our guide for the increase of our doses.

One strict rule we must make, and that is, whenever a general reaction occurs we must make our patients rest in bed until it is over. They must learn to do this of their own accord if treated at home; and if in hospital or sanatorium every sister or nurse must know this absolute rule, and see that it is carried out without waiting for instructions from the doctor or other superior authority. It must be the strict rule of the institution, unquestionably obeyed. If it is faithfully obeyed it will usually be found that any such general reaction will have done good and not harm; that the patient will be found to have made greater progress and to have gained greater tolerance after its occurrence. Why, then, you may say, not aim for it? Well, because it is not entirely without risk. With the excessive general reaction there will always be an excessive inflammatory reaction at the site of disease, and this may cause the disease itself to spread and may cause an excessive amount of auto-tuberculin and even tubercle bacilli to be washed out into the blood, and so give rise to unpleasant symptoms, and even light up fresh disease elsewhere. We must remember, too, that during the period of general reaction the patient is in what is called a "negative phase." His vital powers of resistance are lowered. The opsonins and other antibacterial substances in his blood are less than normal, and so he is less able to resist the spread of the tuberculous disease than he was before. So we can easily see that during a severe or prolonged general reaction harm may occur. There is another practical point, viz., that if the general reaction is excessive, the patient may feel horribly ill, and may, especially if at all excitable or self-willed, make up his mind that he will have no more of the treatment, and so our chances may be gone.

Even without any special general reaction there is always a chance of what is called a secondary reaction occurring. This is a late reaction occurring on the third or fourth day after the injection, and characterised by a rise of temperature. This is due to an excessive focal reaction having stirred up the tuberculous lesion into unwonted activity. So the patient gets an unwelcome dose of his own auto-tuberculin, so great as to cause symptoms. These secondary reactions must be very carefully watched for. If they occur, the next dose of tuber-

culin, then about due, *must* be withheld until all reaction is well over. Otherwise that, plus the dose of his own auto-tuberculin, will be far too much for him, and he will get a violent general reaction, and probably be considerably the worse for it. Such a secondary reaction is always a bad thing; if it does occur, the patient must at once be kept strictly at rest in bed. The best warning of its impending occurrence will be an excessive local skin reaction—remember always local and focal reaction correspond closely.

The interval between the doses is of the greatest importance. If the interval is too short the second dose comes before the patient has got over the reaction due to the previous one—while he is still in the negative phase—and so will knock him down still further. If, on the other hand, we wait too long the immunising response, and with it the tolerance gained, will have waned. We shall then have to start again with the same dose, and will get the same result. So we shall be making no progress.

We ought to give our second dose when the tolerance gained is nearly at its height. Then we must give a bigger dose to make up for that tolerance. If we give just the same dose then, it will be tolerated, will cause no reaction, will practically have no effect whatever. If, however, we give a bigger dose, it will cause a reaction, followed by an immunising response, and a still further rise of tolerance. In this way we can give ever bigger and bigger doses, which is what we want. We see, therefore, that the interval and the increase of dose go together, and must be considered together. In practice it is found that an interval of two or three days works best, and we give our tuberculin twice a week. When we get to large doses the tolerance produced by them lasts longer, and so we can steadily increase the interval.

The next point is, What dose are we to go up to—what is to be our maximum dose? There are many differences of opinion as to this. I think we may accept provisionally the rule given by Drs. Riviere and Moreland, viz.:

(1) That the dose should be increased until at least tenfold the quantity which first caused a reaction has been given, otherwise the tolerance we have established will be insufficient to meet any excessive auto-inoculations, and will not last long enough to be of much avail.

(2) That in ordinary cases we should not go beyond 100 cubic millimetres.

(3) Only to give more than 100 in advanced cases, where symptoms have not improved up to this dose. This means that these patients have been accustomed to get large doses of their own auto-toxins, and have acquired a certain amount of tolerance for such large doses, and will therefore require their tolerance to be raised much higher before it will be of much use to them. So we must get them up to much bigger doses; but practical experience teaches that it is well not to exceed 1000 cubic millimetres, *i. e.* 1 c.c.

(4) Again, if patients cannot be kept under our observation when once the course is at an end, it is well to proceed to much larger doses, so as to give them a degree of tolerance which will be more lasting. Here, again, we should not go beyond 1000 cubic millimetres.

The duration of this tuberculin course, thus roughly sketched out, will vary greatly according to the way in which patients can take it, etc., and according to the maximum dose. Roughly speaking, we may say that it varies from three months upwards; that it should be continued until the maximum dose decided on has been reached.

When this maximum dose has been reached, what is to be done then? Here, again, there is much difference of opinion. Much depends on the condition of the patient. Some recommend that tuberculin be then stopped, and a second similar course be given later on. Personally, my feeling is this: a high degree of tolerance has been gained, enabling the patient to go about and do his work well and remain in good health. We ought, if possible, to maintain this tolerance. This we can do by giving large doses of tuberculin—doses which he can tolerate—just when that tolerance would of itself begin to wane. So we keep him in good condition and able to work, and give Nature time for the ultimate cure of his disease.

I think a good practical rule is that, when we begin to reach large doses, say 10 c.mm., we should gradually increase the interval between our doses. When we have reached our final dose of 100 c.mm., our interval should be one month. We should then continue to give the same dose once a month as long as they continue to improve and for some little time after all tubercle bacilli, all sputum and all other symptoms have disappeared. If at any

time while doing this we meet with difficulty, *i. e.* get reactions, then we should go back to a smaller dose at lesser intervals.

We do not know how long immunity thus procured will be maintained. It will always be well to keep the patients under observation for some two or three years after they have been apparently cured, and to be ready to give them a fresh course of tuberculin should any fresh symptoms or signs of mischief arise.

We now, I think, want some general rules to guide us when giving tuberculin as to whether we are doing good by it or not.

The signs that it is doing good are:

(1) The patient's own feeling of increased well-being, a feeling of fitness, and with it an increased appetite.

(2) The objective signs of—

(i) Increased weight.

(ii) Reduction in pulse-rate, and especially in the variability of pulse-rate on exertion.

(iii) Improvement in the temperature chart, not only lessened fever, but also less daily variations in temperature—a flattening chart.

(iv) Decrease of sputum and of the number of tubercle bacilli contained in it.

(v) The absence of complications.

The signs that tuberculin is doing harm are:

(1) Loss of appetite and nausea.

(2) Mental depression and nervousness and general malaise.

(3) Increase of sputum.

(4) Loss of weight.

(5) A tendency to febrile reactions.

If any of these unfavourable signs make their appearance we must carefully consider the whole case, and be prepared to alter our dosage, or even to give up tuberculin altogether for a time and trust entirely to simpler methods; especially we should search for, and, if possible, remove any adverse conditions. If we decide to recommence our tuberculin we should begin with much smaller doses, and increase the doses much more gradually.

There yet remain for brief consideration—

(1) The treatment of febrile cases.

(2) The question of mixed infection and secondary infection.

(3) The ambulant or out-patient treatment of phthisis.

Febrile cases.—The first question is, Is the fever due to the tubercle bacillus, or to some secondary or mixed infection? If due to the tubercle bacillus the great treatment is absolute *rest*, so as to reduce auto-intoxication as much as possible. That rest should be in the open air.

If, however, rest will not reduce the fever, *i. e.* if our case is one of the *resting febrile* cases—can we get any help from tuberculin? I think we can; but it is difficult. If we can manage to raise *tolerance* beyond the point to which auto-intoxication reaches we shall make the doses of the patient's auto-tuberculin no longer harmful, and the fever will disappear. It *is* difficult. We must be content to begin with very small doses and to increase them very gradually; and we must insist upon our patient resting absolutely; but in many cases it can be done, and when it has been done our patients will continue to improve. It is, I think, best to use T. R. in these cases, as being the least toxic and the mildest of the preparations, and also, being insoluble, it is more slowly absorbed, and so is less liable to cause a strong focal reaction.

We next come to the question of secondary infections and their treatment. We must bear in mind that there are two essentially different conditions—true mixed infections and the so-called attendant secondary infections.

In true mixed infections the other micro-organisms are living in most intimate relation with the tubercle bacillus. If we take the smallest particle of sputum we find both present, and we cannot remove either by repeatedly washing the sputum in running water. This is the true symbiosis. The organisms which are most often found in symbiosis with the tubercle bacilli are streptococcus, pneumococcus, *Micrococcus catarrhalis* and other catarrhal microbes, and staphylococcus. The association with the streptococcus is very bad; each seems to be rendered more virulent by the other; the result is the typical hectic fever. Treatment is apt to be very unsatisfactory and the prognosis is very bad.

Pneumococcus is apt to cause a high continuous temperature and may be very harmful.

Staphylococcus in symbiosis is but little harmful. It is even said to render the tubercle bacillus less virulent.

The other condition is that of the attendant secondary infections. These, I believe, are very

common, and have a great deal to do with the troublesome symptoms of phthisis. The micro-organisms are not in intimate relationship with the tubercle bacilli; they may not get further than the smaller bronchi or bronchioles. These attendant infections respond readily to treatment—either to open-air treatment, especially in the germ-free air of the ocean or the high mountains; or to treatment by continuous inhalations; or by autogenous vaccines. I believe they are chiefly due to the various catarrhal microbes. Their cure, which is fairly easy, is, I believe, one of the chief causes of the great improvement we nearly always get when our patients first go to a sanatorium or to the High Alps. Such improvement is apt to be fallacious, and to lead to disappointment, but it is well worth while, nevertheless, for it greatly helps to restore to our patients the power of fighting their disease. We should not, however, be misled by it, nor beguiled into putting off our tuberculin or other treatment, for it may well be that the tuberculous disease itself is steadily progressing all the time.

If a secondary infection is present and it has been decided to continue tuberculin treatment, we must be very careful to give it in such a way as to avoid reactions. Reaction means temporarily depressing the patient's vitality and resisting powers, and during such period of depression the secondary infection may spread and become more harmful.

It is always best to try to cure the secondary infection first, if it can be done, and here vaccines are found to be of service.

It may be truly stated that these secondary infections are the greatest enemies of our phthisical patients. Probably they are the chief causes of death. If they could be prevented phthisis would be a much less terrible disease, and much more amenable to treatment. The important point is that the true mixed infections can only occur when once the tuberculous mischief has broken down, ulcerated—become "open" as we say. As long as we have only a "closed" tuberculous lesion we can have no true mixed infections. Attendant secondary infections of course may occur, but they are readily curable. As soon as tubercle bacilli appear in the sputum, it means that the case has become an open one, and mixed infection may at any time occur.

That is why it is so important for us, if possible, to diagnose our cases before tubercle bacilli appear in the sputum, and why we are so anxious for our tuberculin tests not to be discarded, but to be perfected and to be used. That is why directly a case becomes an "open" one we should *hurry* the patient off to a sanatorium, so that he may be out of danger of such secondary infection.

That is why it is important to treat all secondary infections as soon as possible, before they have established themselves, and become true mixed infections.

Also why out-patient treatment of phthisis by tuberculin is not always as satisfactory as we could wish, because we cannot avoid the risk of secondary infections.

We must remember that an "open" case treated early and thoroughly may become a "closed" case again. Tubercle bacilli under sanatorium treatment may disappear from the sputum, and all sputum even may cease; then our patients for the time are out of danger of secondary infections; but it is important that their treatment be continued, and a cure if possible be obtained, for they may at any time break down again.

It is stated—and I myself am inclined to believe it from what I have seen—that all closed cases of pulmonary tubercle will get well if treated by tuberculin.

So it comes to this: the important thing is that all cases of pulmonary tuberculosis should be diagnosed early—if possible while they are still "closed" cases. That when diagnosed, treatment should be commenced without delay and be the very best obtainable. That the ideal treatment is treatment in a good sanatorium under skilled medical supervision with tuberculin. That at such a sanatorium there should be some system of carefully graduated and carefully organised manual labour, so that the patients may be turned out fit and strong and able to earn their own living; but that this system of graduated labour should be subsidiary to the treatment by tuberculin.

Lastly, we come to the so-called "ambulant" or out-patient treatment of phthisis by tuberculin. Though sanatorium treatment + tuberculin is by far the best, there are many cases for which it is either not available at all or cannot be long continued.

Cases for "ambulant treatment" fall naturally into four classes:

(1) Early cases of closed phthisis who cannot leave home or give up their work. In these the results are very satisfactory.

(2) Cases who have been to a sanatorium, are better, and are able to return to work, but need the tuberculin treatment to be continued.

(3) Cases who ought to go to a sanatorium, but for various reasons are unable to do so. Here it is only a makeshift treatment.

(4) Advanced cases who are hardly likely to be cured, but for whom tuberculin and the tolerance obtained by its use are very valuable in alleviating symptoms.

The advantages of ambulant tuberculin treatment are these :

(*a*) It enables the patient to remain at home, and often to continue to earn his livelihood. If cured by it, as he has remained at work and under his normal conditions of life, the cure should be lasting.

(*b*) A patient can continue specific treatment when he has left the sanatorium and is at work again ; or, on the other hand, when the disease has become so chronic that no sanatorium would accept him as a patient, and there is no other help available.

It is therefore most essential that such ambulant treatment for phthisis should be provided. It could be carried out in the out-patient departments of our hospitals or in special dispensaries. In either case it should be thoroughly organised, and every help available should be made use of. There should be nurses to instruct the patients how to take their temperatures and to keep those records which are necessary for skilful scientific treatment, and to visit the patients at their homes, give advice on matters of domestic hygiene and on all questions of diet, rest, exercise, etc. Such nurses will need great tact, devotion, and skill. They should be chosen from among our best and best-trained nurses, and be well paid.

The help of the C.O.S. and other charitable agencies will be brought in, and, still more important, that of our public health authorities ; everything possible must be done to improve the patient's surroundings and conditions of life, and to prevent the risk of his infecting those around him.

To enable the ambulant treatment to be both successful and safe there is one thing greatly needed, namely, a hospital for acute, *i. e.* febrile cases. All phthisical patients, and especially patients who continue at work while undergoing treatment, are liable at times to febrile attacks. They should then be put to bed at once and nursed with as much care as if they were cases of typhoid fever. Often this cannot be done at their own homes, and is only possible if they can go to a hospital specially organised for the work. Such a hospital Dr. Fraser has provided at Portsmouth, and such a hospital the Sanatorium Benefit ought to provide in every centre.

Such is the work before us in the treatment of phthisis. It is work which ought to be done, and which, sooner or later, will be done. Moreover it is work full of hope as well as full of interest. It is of the utmost importance that it should be done by the right people. Now there is no question to my mind that the right people to do it are the medical men in practice throughout the country. It is our work, and we can do it. If we wish to do it—and I hope we all do—we must make ourselves thoroughly acquainted with modern methods of treatment, and know and master their difficulties and be enthusiastic about the possibilities.

I fully recognise that the work of diagnosis and treatment is only a part of a far more important work—the prevention and ultimate abolition of tuberculous disease. That is the work for our health authorities, our county councils, our Local Government Board to do. *Our* first duty as practitioners of the healing art is to do our best for our individual patients, and I believe that if we do this skilfully and thoroughly we shall be doing a great deal to help those authorities in their work also.

December 9th, 1912.

Actinomycosis in Tuberculous Subjects.—Slattery cites three instances of actinomycosis occurring in patients with positive reactions to tuberculin used in diagnosis. All were looked on for a while as cases of active tuberculosis with foci in the neck glands or in the lungs. One with tuberculous adenitis had been considered suitable for a tuberculin course. But in no one was the acute process due to the tuberculous infection. They were met with at long intervals in different institutions. In one case the examination of discharges showed streptothrix.—*Journ. A. M. A.*, vol. lix, No. 20.

Artificial Pneumothorax in Pulmonary Tuberculosis.—To many previously considered hopeless advanced cases, Otis says, artificial pneumothorax, produced by the injection of nitrogen gas, offers chance of arrest, and the increasing number of reported successful cases by men of the highest authority, and extending over a considerable number of years, has fully attested its value and established this treatment on the firm basis of successful experience. The procedure is a simple one, so far as any operative skill is concerned, but it requires careful manipulation in order to get in between the two layers of the pleura. If firm pleural adhesions exist the lung cannot obviously be compressed, but it is surprising how infrequently this happens, even in very extensive disease. Not only in advanced tuberculosis, but also in abscess of the lung, bronchiectasis, and dangerous hæmoptysis is the operation applicable. In performing the operation, a very careful determination of the exact condition of the diseased lung is made, and in every case the Röntgen ray is used, particularly to ascertain whether or not pleural adhesions exist. Of course, it is evident that the other lung must, for the most part, be able to perform its function; not necessarily that its integrity should be complete, for there may be, and often is, a small amount of disease at the apex. Curiously and fortunately the artificial pneumothorax often exercises a beneficial influence on the diseased apex of the other lung, if it exists, and that, also, becomes arrested, the cause of which has been variously explained. The conditions, then, which warrant a recourse to artificial pneumothorax are an advanced or advancing case of pulmonary tuberculosis which does not yield to the ordinary treatment, the disease being principally unilateral. After a careful physical and Röntgen-ray examination, the spot which appears to be free from adhesions is selected, and, by careful manipulation, a needle similar to an aspirating needle is inserted between the two layers of the pleura, and through it nitrogen gas is slowly injected under slight pressure. Only a certain amount of the gas is allowed to flow in at the first attempt, depending on individual conditions, and the procedure is repeated, if all goes well, at frequent intervals until the lung is tightly compressed, as shown by the physical examination and a Röntgen-ray picture. When this has been accomplished, the subsequent injections are at longer intervals, sufficient to maintain the compression.—*Journ. A. M. A.*, vol. lix, No. 20.

Correction of Nasal Deformity.—The septum in Glogau's case was prepared as for submucous resection by applying a 20 per cent. cocaine solution and a 1 to 5000 epinephrin solution, with cotton pledgets to the Schneiderian membrane. The usual incision was then made on the right side, but the knife was carried right through the cartilage towards the mucous lining of the left side, without, however, injuring the latter. The cartilage was then elevated from the mucous lining on the left side, and by means of a strong pair of scissors an incision was made at its upper and lower margins. The cartilage flap, covered by the mucous lining of the right side, was thus attached at its posterior part only. The nostrils were now plugged with a piece of cotton, and the back of the nose cleaned aseptically. A 0·5 per cent. cocaine solution, containing a few drops of epinephrin, was then injected hypodermically at the middle of the nose toward the tip. A transverse incision of about 15 mm. was then made at the lower end of the nasal bones. Through this incision, by means of a knife, the subcutaneous tissue was separated along the back of the nose down to the tip. A sharp spoon was then introduced into this pocket and the cartilage at the tip of the nose curetted. The external wound was then covered by a piece of gauze. The cartilaginous flap was now severed from its posterior attachment and put into a physiologic salt solution. By means of a Bier's skin-grafting knife the superficial epithelial layers of the mucosa were then removed, and the cartilage, covered by its perichondrium, shaped by means of a pair of scissors to fit into the deformity. The cartilage was then inserted into the deformity, and the transverse incision closed. The wound was covered by a small piece of iodoform gauze and adhesive plaster. The mucous lining over the bony part of the deviated septum was then elevated on both sides, and the bony deviation removed by submucous saws. The septal mucous lining of the left side remained intact, while on the right side there was a loss of substance corresponding to the piece of cartilage removed with its right-sided mucoperichondrium. The nasal cavities were then painted with a 10 per cent. oily solution of iodine and packed in the usual way. The intra-nasal packing was removed after forty-eight hours, and the dressing of the external wound was changed every day. After the fourth day the ligature began to suppurate, and the wound opened again. Healing by granulation took place. The inserted cartilage became permanently attached, and the deformity is now considered to be corrected.—*Journ. A. M. A.*, vol. lix, No. 21.

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THE SELECTION OF CASES OF PULMONARY TUBERCULOSIS FOR SANATORIUM TREATMENT.*

By J. EDWARD SQUIRE, C.B., M.D., F.R.C.P.

THE selection of cases for sanatorium treatment presents a somewhat complex problem. Whatever may be said of the general results of sanatorium treatment as a curative agency, there can be no doubt that the great majority of tuberculous individuals derive benefit from residence in a well-equipped and well-organised institution in the country, where fresh air, good food and medical supervision can be obtained. At the same time there is not accommodation for all who would like to avail themselves of these advantages, nor is it possible to arrange and staff these sanatoria so that the early and the advanced cases can be received in the same institution. Selection of cases is, therefore, essential.

In the case of private sanatoria where high fees are charged, the first selection is provided by the monetary requirements, and the applicants for admission are rarely in excess of the accommodation. If the applicants are able and willing to pay for nursing and the other requirements of a bed-ridden patient, even those in the last stage of the disease may be accommodated. But in such institutions, also, though a greater latitude of selection may be allowed, selection on medical grounds is still desirable. Patients who may have a good chance of recovery are likely to be discouraged by the presence of hopeless or dying cases unless these form but a very small proportion of the inmates of the sanatorium. The companionship of those who are progressing favourably

* A lecture given at the Medical Graduates' College and Polyclinic.

encourages those who may not yet be conscious of improvement in themselves. There can be no doubt as to the desirability of keeping advanced and hopeless cases apart from those who, though attacked by tuberculosis, have yet a fair expectation of recovery.

But tuberculosis is pre-eminently a disease of the poorer members of the community. By far the greater number of consumptives are unable to pay large fees for sanatorium accommodation, and many are unable to pay any fee at all, even if they can afford to give up their work to enter a sanatorium. The large proportion of consumptives are thus dependent on institutions built and maintained wholly or in part by the charitable or by the State. Thus it happens that the beds available are lamentably deficient for the number of applicants, and selection is a necessity.

Since only a proportion of the sufferers from consumption can be accommodated, it is of importance that the beds available should be utilised to the greatest advantage. How, then, may a sanatorium bed be made to provide the greatest benefit? Obviously we must consider here the greatest benefit to the greatest number. We cannot afford to allow our judgment to be diverted by our sympathy for the individual consumptive. This natural tendency to be moved by the claims on our sympathy of each applicant who presents himself constitutes one of the great difficulties of the examiner who is entrusted with the selection of cases for admission to a sanatorium for poorer consumptives. There may be twenty beds available and one hundred applicants. Each one would be better off in the institution than in his own home—he would be living with little or no expense to himself or his friends, well fed, and well cared for. In almost every case his condition would be improved by a few weeks' residence in the institution. In many cases his family would benefit by the removal of a source of infection from amongst them. In each case, probably, the doctor who has seen him at home has told him that he is a fit case for sanatorium treatment, and that such treatment would be of benefit to him. It is an unenviable task to have to reject a large proportion of these people, knowing what an advantage it would be to each and every one if they could have a few weeks' sanatorium treatment. True, the benefit would in many cases be but temporary, but one would gladly afford them even this if it were possible.

Since, however, only a portion of the number can be accommodated, on what grounds shall we base our selection? Two main principles present themselves at once for consideration. In the first place, permanent arrest of the disease, allowing the individual to take his place again amongst the wage-earners with every expectation of continued health, is of more value than temporary return to work with the probability of a breakdown later on. So also the admission of a case which offers the probability of arrest with a few weeks' treatment is economically more advantageous than that of a patient in whom arrest of the disease is likely to be a matter of many months. Thus, in our selection, we should attach great importance to the probability of permanent arrest within a reasonable time. Secondly, the consumptive who is expectorating sputum laden with the germs of the disease is a constant source of danger to others unless he knows and observes the precautions necessary to avoid the spread of infection. In such cases, though the cure of the affected individual may be unlikely, there is obvious advantage in affording him practical instruction in the precautions necessary for preventing infection of others.

Sanatoria may, therefore, be usefully employed as curative and as preventive institutions. The proportion of beds allocated to each of these branches of the work must depend to some extent upon the nature of the cases applying for admission. If there were institutions for the reception of advanced consumptives, with adequate accommodation, these would be the proper places to receive the cases whose condition precluded the hope of permanent arrest, but who needed instruction in preventive precautions. At present, though a majority of applicants for admission to special hospitals and sanatoria come under this category; there is practically no institution except the sanatorium where they can be received for such instruction.

We must, therefore, allow that under existing circumstances a certain proportion of cases must be admitted to the sanatorium for instructional purposes. These cases will be admitted for a limited period; four weeks may be taken as a suitable term. Those admitted for cure will remain

till arrest is obtained, or until it becomes evident that there is no immediate prospect of such a favourable result. The essential work of selection is thus reduced to the recognition of the kind of case which offers a probability of arrest within a comparatively short period, such as three to six months. We must remember that when arrest is achieved within such period, it will require a still longer period of care to consolidate the "cure" or give permanence to the arrested condition, though this longer period need not be spent in a sanatorium, and may be usefully employed in work. From a somewhat extensive experience of examining applicants for admission to sanatoria I gather that there are very indefinite ideas as to the kind of case which may be expected to become arrested after a few months' sanatorium treatment. Cases are sent up as "early tuberculosis of the lung with few physical signs" when there is evidence of softening in both lungs; others, similarly described, present the physical signs of general fibrosis of the lung. Such mistakes may be due to misinterpretation of the physical signs in the chest, or they may easily occur if undue reliance is placed on physical examination without the corrective of the personal history of the patient. Here it may be well to note that a reliable personal history is not always easy to obtain; many persons find a difficulty in giving a lucid history of an illness, and amongst the poorer patients this difficulty is increased by their want of facility of verbal expression. A certain amount of cross-examination is generally necessary. A patient will inform you that there was nothing whatever the matter until he got a chill a month or two back. A little questioning may elicit the fact that twelve months or so before there was hæmoptysis followed by a few months' illness. An interval of comparative well-being seems to the patient to justify the description of the present breakdown as a fresh and distinct illness.

I must emphasise the importance of looking upon any attack of blood-spitting which cannot be definitely explained as indicating probable tuberculosis of the lung. In the absence of heart disease it is unwise to accept over-exertion, lifting weights, or hot weather as sufficient explanation of such occurrence. A healthy lung does not bleed from such causes as the above; a lung affected by early tuberculosis may do so and thus give timely warning of unsuspected disease. Bleeding from the throat or gums may of course occur, but these should not be accepted as the source of the blood unless there is some corroborative evidence.

I have frequently had to reject applicants as too advanced for admission in whom a warning hæmoptysis some months or years previously had been disregarded or attributed to some comparatively trivial cause, because there were then no recognisable signs in the chest. It cannot be too strongly insisted upon that the physical signs of *early* tuberculosis in the lung are slight, and, to the inexperienced, indefinite. They are unlikely to be detected at all unless the chest is completely bared for examination—they cannot be recognised through the clothing.

When such signs—deficient respiratory expansion, slight local dulness, or weak, rough breath-sounds—are noticed, their significance is apparent when they occur in conjunction with symptoms of lassitude, easily induced fatigue, loss of weight, occasional rise of temperature and other signs of ill-health, each of which by itself might be considered trivial. If there has been a slight blood-spitting as well no hesitation should be felt in putting the patient under treatment without delay. We need not expect to find tubercle bacilli in the sputum; there may be no expectoration and no cough. Sputum containing bacilli is not likely to be expectorated until a later stage when softening has taken place. To wait till microscopical examination of sputum confirms the diagnosis of tuberculosis of the lungs is to put off treatment until the prospect of arrest is dangerously diminished.

But we must not confine our attention merely to the stage of the disease in selecting cases for curative treatment; we should be guided also by the type of the disease and the general condition of the patient. Tuberculosis, when it affects the lungs, does not advance with the same rapidity in all cases nor in the same manner. The classification of a case as an "early case" of pulmonary tuberculosis is not purely an estimate of the probable time during which the disease has been present, nor even of the time during which it has been active.

I have seen "advanced" cases with a history of three months' illness and "early" cases with a twelve months' history. Such differences in the rate of progress may be due to the dose and virulence of

the original infection or to the resisting power of the individual, which may be local or general. The resultant of these forces varies with the relative potency of the two factors, and we may recognise marked differences in the course of the disease according as the one or the other of these factors predominates. Thus we come to recognise types of pulmonary tuberculosis which are likely to follow different courses, and our prognosis will be influenced by our classification of any case according to the type of disease in which we place it. I cannot here attempt to set down a classification of types with their distinguishing characteristics. It is largely a matter of experience which guides the giving of a prognosis in each individual case. I will merely take as an illustrative example two different classes of cases which tend to different results. It is within the experience of most of us that in one individual, tuberculosis, when it attacks the lung, tends to remain limited to the apex, the activity of the disease manifesting itself in the softening and excavation of a limited area of lung-tissue. The force of the disease is, as it were, concentrated, and its effects are destructive but localised; there is little tendency to extension, but comparatively rapid progress through the stages of infiltration, disintegration and excavation in the "storm area." In another case we find the tendency of the disease is to extend over an ever-increasing area, remaining apparently at the stage of infiltration with little or no tendency to softening. The disease exerts its force in the extension of its area rather than in the complete destruction of the part first invaded. There is diffusion instead of concentration, and the physical signs at any one part of the affected area would suggest a less virulent form of disease than in the first kind of case. Yet this creeping type of the disease is much less favourable to recovery than the type where the virulence of the disease is concentrated. Much may be gathered as to the resisting power of the individual and the consequent chances of overcoming the disease by a consideration of the physique, age, surroundings and family history of the patient. We have, most of us, had impressed upon us the characteristics of the "phthisical chest," but few of the candidates for sanatorium treatment have such a chest. The broad-shouldered, deep-chested individual who has become infected from an overwhelming dose or from continued infection for a prolonged period has every chance of recovery, whilst an undersized, small-chested man with small expansion in a less advanced stage of disease would be a less favourable subject for admission. Fat and flabby individuals, though they may look big and strong, are, in my experience, unfavourable cases. Youth is a strong point in the patient's favour. A young adolescent, with active and comparatively advanced disease, may yet justify admission to a sanatorium, when a less active case of thirty-five years of age would be rejected. Few cases over forty or forty-five years of age would justify selection if their admission would keep out, or delay, the admission of more hopeful cases. These older individuals, when affected with pulmonary tuberculosis, generally show little tendency to arrest of the disease, though it may advance but slowly. Children under fourteen years of age often take a long time, even three or four years, before the disease becomes arrested, though it remains in a semi-quiescent condition. After this age they often improve rapidly under sanatorium treatment. A history of tuberculosis in the family suggests a constitutional deficiency of resisting power against the disease and renders the prognosis less favourable. I do not attach much importance to a family history of tuberculosis in arriving at a diagnosis. We know that about one third of those who become tuberculous are the offspring of tuberculous parents. On the other hand, there is, I think, little doubt that the family history is important in prognosis. The offspring of tuberculous parents are more likely to suffer severely from tuberculosis if they become infected than are those with a clear family history. I have recently been going through a large number of death claims for life assurance, and I find that the proportion of deaths from tuberculosis amongst those who show a family history of the disease is more than twice as great as amongst those with no such history (30·82 per cent. as against 13·52 per cent. in 2500 cases). We must remember that these are selected lives.

It is perhaps advisable to refer to the tuberculin test as an element in the selection of cases. We come across cases recommended for sanatorium treatment on the strength of a positive reaction to tuberculin, whether by Koch's original method (subcutaneous injection) or by v. Pirquet's method (skin reaction), or Calmette's (ophthalmic) test. If we were to accept a reaction to tuberculin—without

any other signs of disease—as sufficient to justify admission to a sanatorium, we might easily fill such institutions with individuals who might well be at work. No doubt any of us would benefit by three months' sanatorium treatment, but it is a serious matter to make a wage-earner give up three months or more of work unnecessarily. In doubtful cases—that is in cases where we have a suspicion of tuberculosis but are unable to detect definite physical signs—all aids to diagnosis should be carefully employed. The temperature and pulse should be recorded and watched, the sputum, if there be any, should be microscopically examined, and the individual may be tested with tuberculin. The evidence derived from each of these must be given due weight. The sum total of all the evidence procurable may justify a diagnosis of tuberculosis, but no single point in the evidence—unless it be the presence of tubercle bacilli in the sputum—should be relied upon alone. I have recently seen a case which I admitted for observation, in whom the symptoms and physical signs were very slight and indefinite, where Koch's old tuberculin produced no reaction, but after the injection tubercle bacilli were found for the first time in the expectoration. In this case at least the tuberculin test—though it produced no reaction—provided certain evidence of tuberculosis where it had only been suspected.

I would again emphasise the well-known fact that the probability of a satisfactory result from sanatorium treatment depends largely upon the selection of "early" cases, and that the detection of the disease in a curable condition demands careful examination, and a due appreciation of slight deviations from the normal physical signs in the chest. I cannot too strongly urge upon those who have to examine the chest for signs of tuberculosis—and this includes practically all who are in practice—that they should take every opportunity of examining healthy chests.

Life assurance work is very useful in this connection if the examinations are properly conducted.

Without being familiar with the physical signs of the healthy chest, it is impossible to recognise slight deviations from the normal.

The left side of the chest generally expands somewhat less than the right, and the respiratory movement is thus less at the left apex than on the right. The percussion note is often (in about 50 per cent. of healthy chests) higher pitched and "flatter" on the right, whilst the breath-sounds are louder on the right and the vocal resonance and fremitus more marked than over the left apex.*

Thus relatively deficient reasonance has more significance at the left apex than at the right, and relatively weaker breath-sounds on the right as compared with the left are at once suggestive of disease.

But though we give preference to the early cases, we must not exclude all who cannot be thus classed.

Persons who have so far recovered from an attack of pulmonary tuberculosis as to have remained free from symptoms for several years may for some reason get a fresh outbreak in the site of the old lesion or in an entirely distinct part of the same or in the opposite lung. This fresh outbreak of active tuberculosis may be treated as an early case, though the history of the disease as given by the patient extends back for several years. Such cases frequently do well under sanatorium treatment.

These cases are, however, quite distinct from the "chronic" cases in which the disease has remained active, though subacute, for several years. These are unsuitable for admission to a sanatorium.

The probabilities of arrest in pulmonary tuberculosis vary with the number of lobes affected. In the medical report for 1908 of the Mount Vernon Consumption Hospital, it is noted with reference to the sanatorium at Northwood that of fifty-five cases in which arrest of the disease occurred, 89 per cent. had only one lobe of the lung affected whilst the remainder had two lobes—on the same or on opposite sides—affected. No cases in which three, four or five lobes were affected became arrested.†

A sanatorium is hardly the most suitable place for the man who has suffered from consumption for five or six years with consequent fibrosis of his lungs. There may still be evidences of active (subacute) tuberculosis, but in such a case as this such smouldering mischief will remain in spite of sanatorium treatment. There may be general weakness, shortness of breath, and cough; there may be tubercle bacilli in the sputum. But

* "The Physiological Differences between the Two Sides of the Chest on Physical Examination," 'Brit. Med. Journ.,' 1903, i, pp. 1198–2200.

† *Vide* "Prognosis in Pulmonary Tuberculosis," 'The Hospital,' June 19th, 1909, p. 306.

though these facts show the necessity for treatment, such treatment as is required can be carried out at home with as much success as would be achieved in a sanatorium. If the home conditions —or want of a home—make home treatment impossible, some hospital is more suitable than a sanatorium..

Consumptives with much bronchitis or who are liable to bronchitis rarely do well in the open-air conditions of a sanatorium unless the weather is dry. It is, therefore, unwise to admit such cases except in the summer months, and then only in a dry season.

There is a large class of cases in which a person, having suffered for several years from winter cough, gets tuberculosis of one apex superadded to his chronic bronchitis. Such cases very rarely do well in a sanatorium.

It is not an uncommon experience to find that a person who may have recovered from an attack of pulmonary tuberculosis—it may be years previously —presents himself for admission to a sanatorium.

The permanent changes in the lung which have resulted from the disease, scars or fibroid overgrowth, with consequent loss of elasticity and possibly emphysema, produced physical signs in the chest which may be mistaken for commencing disease. Further investigation would correct this impression. Perhaps the fear of losing valuable time induces the doctor to tell the individual to try and get into a sanatorium without delay, or the person himself may take advantage of his damaged lung to seek a few weeks' rest in the comfortable surroundings of sanatorium life. I have heard of enterprising individuals taking advantage of a long healed lesion in the lung to obtain admission to a sanatorium, draw sick pay from one or more clubs whilst living free in the institution for a few weeks, and thus obtain sufficient money for a further holiday on their own account. Let us hope this is merely a fable ; I have, however, come across cases which suggest that the fable may be at least founded on facts. Sanatoria are obviously not maintained to harbour such cases.

Cases of acute tuberculosis, with high temperature, are better kept at home in bed till the temperature is reduced before they are sent to a sanatorium. The journey itself is likely to upset them, and since they must remain in bed whilst the temperature is high, there is no object in sending them away if they can obtain fresh air and attention at home. When they are able to get up they will be more likely to gain the full advantage of the sanatorium *régime*. If the home conditions are unfavourable it may be wise to risk the journey and utilise the sanatorium as a means of getting them away from unhealthy surroundings.

Pulmonary tuberculosis complicated with disease in other organs may be quite unsuitable for admission, and the complication may require to be first treated in order that the patient may gain the greatest benefit from the sanatorium. For instance, I have recently seen a case of early pulmonary tuberculosis with a tuberculous testicle which should be removed if the lung mischief is to have any chance of recovery.

In this connection we may note the advisability of having bad teeth attended to before admission. Pyorrhœa alveolaris is far from uncommon in applicants for sanatorium admission. Such a condition prevents the individual from gaining the advantage from the sanatorium which he should do.

How far we are justified in admitting cases with marked laryngeal tuberculosis into a sanatorium must depend to some extent on the facilities for laryngeal treatment in the institution. No doubt laryngeal cases benefit from sanatorium conditions but they generally require special treatment also.

It would be a good thing if there were special sanatoria for laryngeal cases, with expert laryngologists as medical superindendents, and all necessary appliances for treatment of the throat conditions. As a rule cases with extensive laryngeal tuberculosis are not suitable for admission to an ordinary sanatorium. In my experience cases in which the lung and larynx become affected almost simultaneously, *i.e.* cases of *early* pulmonary tuberculosis complicated with tuberculosis of the larynx do not, as a rule, do well.

In all cases of tuberculosis a consideration of the utmost importance, which cannot be overlooked, is the condition of the patient's home. Treatment is unlikely to be successful, or at least can hardly be so successful, when the patient is living under conditions which are unfortunately far from uncommon in the homes of the poor. Dirt and overcrowding in ill-ventilated rooms, and possibly insufficient nourishment, will seriously affect the success of any treatment. The sanatorium affords an environment in which the individual has the

best chance of benefiting from treatment, whether by rest, food or drugs—this is, indeed, the essential purpose of the sanatorium. A patient living in a healthy home may be successfully treated without removal to a sanatorium, whereas another whose home conditions are unsatisfactory should be treated elsewhere.

This consideration, however, is not in itself a justification for admission to a sanatorium. If the case is one which offers the expectation of permanent benefit from treatment, removal to a sanatorium when the home conditions are unfavourable is clearly desirable. On the other hand, the advanced and incurable cases, though removal from unhygenic surroundings is equally desirable, should be admitted into special institutions for such cases rather than into a sanatorium.

It will be evident from a consideration of the various points touched upon above, that the selection of cases for sanatorium admission requires not only experience in the examination of the chest, but some practical experience of the treatment of patients in a sanatorium. By watching the progress of patients in various stages of the disease and of different types we obtain some knowledge of the kinds of cases which may be expected to do well and those who are less likely to benefit.

December 16th, 1912.

Intra-venous Injections.—Galli describes the technique which is in routine use at Baccelli's clinic at Rome, as the Italians apply this method frequently in treating tetanus, sepsis, malaria, etc. The sphygmograph cuff is very convenient for the purpose; the radial pulse must not be abolished, only the systolic pressure. This permits the arterial blood to flow into the arm while the reflux in the vein is arrested. The syringe is filled to four fifths with the fluid and the needle is inserted until it runs parallel to the course of the blood in the vein. Withdrawing the piston permits a little blood to enter the syringe if it is really in the vein. The contents of the syringe then are slowly injected and the piston is withdrawn a trifle again just as the pressure in the cuff is released. In his extensive experience with the method he has never witnessed the slightest untoward by-effects or pain from the injections.—*Journ. A.M.A.*, vol. lix, No. 21.

LECTURES ON NEUROLOGY.

Delivered to Post-Graduates at St. Bartholomew's Hospital,

By C. M. HINDS HOWELL, M.D., F.R.C.P.,
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LECTURE I.

EXAMINATION OF CASES OF NERVOUS DISEASE.

BEFORE demonstrating to you the two cases which I have brought to-day to illustrate methods of examination of the nervous system I want to say a few words introductory to the subject.

First I want to emphasise the necessity of always making a *complete* examination of the central nervous system in every case which presents, or seems to present, a simple isolated nervous lesion. Rules that hold good in other branches of medicine cannot be disregarded here, and it is through want of making a thorough examination that mistakes in diagnosis, or the failure to make a diagnosis at all arise. If a patient comes to you complaining of trouble with a joint you examine *all* his joints; or if a group of glands are enlarged the whole lymphatic system should be examined. It is the same with diseases of the nervous system.

Perhaps in no other branch of medicine is the history given by the patient of more importance. As a rule in heart disease objective examination enables an accurate diagnosis to be made, but this does not hold good in many cases of nervous disease, so that the history of the case must be thoroughly gone into.

Leading questions have often to be used, though with discretion. The patient may have paid little or no attention to symptoms which are really of the first importance in diagnosis, as, for instance, the occurrence of gastric disturbance, or pains in the limbs with a swollen knee, which turns out to be a Charcot joint. Again, temporary visual disturbance or precipitancy in micturition possibly occur months before more definite symptoms of disseminate sclerosis begin to make their appearance.

Very important is the *family history*; this is especially true when we are dealing with functional disorders or neuroses of various kinds. Consanguinity of the parents, mental disorders, alco-

holism, epilepsy, asthma, and so on should be inquired for, as their occurrence undoubtedly influences the prognosis in certain cases very materially.

The previous health of the patient is another important point to be considered. The question of syphilis at once occurs to you, taking as it does such a heavy toll from the nervous system. In this connection too much reliance must not be placed either on a positive or negative history, as both may be misleading. Much more important from the diagnostic point of view are:

(1) The Wassermann test.

(2) The condition of the cerebro-spinal fluid, of which the latter is by far the most reliable, and to the consideration of which we shall return in a later lecture. Apart from the question of syphilis the early history is of importance, as it may give evidence of an unstable nervous system of congenital origin. Such occurrences as "teething convulsions," nocturnal enuresis, chorea, and so on must all be considered, say, in giving a prognosis in any given case of epilepsy, or some other neurosis. So also influenza, the exanthemata, tuberculosis in one form or another, and trauma may all sow the seeds for later disturbances of the nervous system.

Whilst talking to the patient much may be gathered from his appearance and speech. The mask of paralysis agitans, the tremor and hesitating speech in general paralysis of the insane, for instance. The intelligence, memory, emotional character, and so on must also be tactfully investigated.

Stigmata of degeneration should also be looked for: such are to be found in the shape of the head, in the size, form, and symmetry of the ears, and the shape of the jaw and palate. The lower jaw may be under-hung, with projecting teeth, and the high-arched gothic palate is of course characteristic.

By following these lines in your preliminary investigation you will have arrived at some idea as to the psychology and nervous temperament of your patient—points which in a great many instances are as important in connection with the case as the more gross signs of organic disease which may be present.

Having cleared the ground with these preliminaries the routine examination of the patient should be performed. Such an examination must of course include *all* the different systems, but it is with the nervous system that we are particularly concerned for the moment.

The special senses should be first investigated. Disorders of smell and taste are seldom sought for in the ordinary examination, and yet their presence on occasion furnishes most valuable information.

The olfactory lobes you will remember lie beneath the frontal lobes, along their orbital surfaces, and are connected in a complex manner with the brain, the ultimate representation of smell, however, being chiefly centred in the hippocarpal convolution.

The sense of smell should be tested by such familiar odours as peppermint, cloves, lavender, etc. Ammonia or similar substances which may stimulate the fifth nerve should be avoided. If a defect of smell is detected on one side, careful examination of the nose should be made to exclude any local abnormality which may exist. Disorders of smell are met with both in organic and functional nervous disease.

(*a*) *In organic disease.*—(1) Tumours, particularly endotheliomata which are situated on the lower surface of the frontal lobe, and, being hard, compress the olfactory nerves, often cause loss of smell (anosmia). If the tumour is a glioma it is less likely to do so, as this variety is soft and infiltrates the cerebral tissue, but the increase in pressure which it produces *may* cause alteration in the sense of smell. (2) Tumours of the temporo-sphenoidal lobe when situated towards its anterior and mesial surface sometimes cause disturbances, usually of a subjective character, in the sense of smell. Such tumours are often associated with fits, and an olfactory aura may precede the attack. Tumours in this region are sometimes of slow growth, and I have seen several cases where for years (the longest seven years) the only symptom exhibited by the patient was epilepsy, the fit being often preceded by an olfactory aura. It is interesting to note that this aura is usually an unpleasant odour. I don't want to give you the idea that every case of fits preceded by an olfactory aura is due to tumour, but I want you to remember the possibility of its being so, and to keep a careful watch on the optic discs in case papillœdema develops.

(*b*) *Functional disorders.*—We have already referred to idiopathic epilepsy, but apart from this anosmia may occur in hysteria. I have a patient under my care at the Great Northern Hospital in

whom there has been hysterical loss of *all* the special senses on the right side. I hope to be able to show her to you on a later occasion.

With regard to *taste*, the anterior two thirds of the tongue are supplied by the fifth and chorda tympani nerves, the posterior one third, and the circumvallate papillæ by the ninth:

Taste is tested by strong solutions of sugar and sodium chloride, by dilute citric acid, and a dilute solution of quinine. These are applied to the surface of the protruded tongue by a glass rod, and if the taste is recognised the patient writes down the name "sweet," "salt," "sour," "bitter," as the case may be, without withdrawing the tongue. After each test the mouth should be rinsed, and it is well to test with quinine last as its stimulus is more permanent than any of the others. Loss of taste may occur in disease of any of the nerves mentioned, and is associated therefore with some cases of facial palsy of a peripheral type when the lesion occurs in the temporal bone within the Fallopian canal. It also occurs as an hysterical manifestation.

Vision.—Three points require examination:

(1) Visual acuity, which is investigated with the ordinary test-types. It is important to remember that optic neuritis may exist in its early stages without any reduction of visual acuity, and so, though the vision remains perfect, the examination of the fundus oculi must never be omitted.

(2) Visual fields: To investigate them accurately the perimeter must be employed, and it is important to test the colour-fields as well as the field for white. This method often gives valuable information, and may afford the only clue as to the position of a tumour or other intra-cranial lesion. Also the discovery of central scotomata, indicative of retro-bulbar neuritis, will be possible, and may confirm the diagnosis of tobacco poisoning (central scotoma for red), or of early disseminate sclerosis and so on.

The extent of the visual fields can be investigated roughly without the perimeter by a very simple method, and this should always be done. If hemianopia is present it is important to decide whether the lesion is in the optic chiasma, optic tracts, or further back in the cerebral visual paths.

To determine this use is made of Wernicke's pupil reaction. This is done by throwing a light in a dark room onto the blind half retina, whilst the patient looks straight in front of him. If the pupils do *not* react to light under such conditions the probability is that the lesion involves the chiasma or tracts, whilst if reaction takes place the lesion is probably situated in the intra-cerebral visual paths or visual cortex.

(3) The fundus: In every case of nervous disease the fundus should be examined, and particularly is this important in every case in which either convulsions or headaches are complained of. The presence or absence of papilloedema is, of course, a point of paramount importance, but in addition to this much information can be obtained from the appearance of the vessels, *i. e.* whether there is venous congestion, or "silver wire" arteries indicative of arterio-sclerosis, hæmorrhages or atrophic patches such as occur in renal disease, or tubercles in the choroid. Time will not permit of my doing more than mention such conditions, and one must now pass on to the last special sense to be examined, namely, *hearing*. The eighth nerve you will remember consists of two parts: (*a*) the auditory or cochlear division, (*b*) the vestibular.

(*a*) Auditory division concerned with hearing may be tested by watch, tuning-fork, or whispered voice.

If deafness is present the following tests should be applied to determine whether the condition is due to nerve- or middle-ear disease.

(1) *Rinne's test.*—Apply a tuning-fork to the normal mastoid process; when it is no longer heard apply to external auditory meatus and the sound in a normal ear will still be heard—that is to say, air-conduction is normally better than bone. This is a positive Rinne's test. If there is deafness with positive Rinne the lesion is probably in the nerve, if negative in the middle ear (negative Rinne means bone-conduction better than air).

(2) *Weber's test.*—The tuning-fork is placed on the forehead. If heard in deaf ear best disease of middle ear, if in normal ear probably nerve-deafness present.

Vestibular nerve.—This may be investigated at the same time. If diseased the patient usually complains of unsteadiness owing to great giddiness and of noises in the ear.

There may also be vomiting in severe cases (Ménière's disease) and nystagmus.

The tests for the vestibular nerve are rather complicated, but their general plan can be indicated. For further details you must consult the aurists.

If the semi-circular canals (supplied by vestibular

nerve) are *normal*, you can stimulate these in the human being.

Response to stimulation is shown by jerking (nystagmoid) movements of the eyes. The reason for this is that the vestibular nerve is closely connected within the brain stem through Deiter's nucleus with the nuclei of the third, fourth, sixth, cranial nerves. Giddiness and a sense of rotation will also be produced. If the labyrinth is diseased these effects will not be produced. The stimulus is applied to the patient in one of three ways (Bárány).

(1) Rotation on a music stool or special table with the head in various positions. On cessation of rotation and deviation of eyes to side from which rotation has been made nystagmus should occur in normal persons.

(2) Irrigation of ear with hot or cold water should produce nystagmus. It may also produce vomiting.

(3) Passage of a galvanic current through external auditory meatus may again produce nystagmus. If it is impossible to produce nystagmus by any of these methods the labyrinth is certainly at fault.

Time will only permit of my making a very few remarks as to the examination of the rest of the cranial nerves which must be undertaken next.

Third, fourth, sixth (oculo-motor group): The range of movement of eyeball must be noted both in conjugate movements, and the action of individual muscles investigated. The condition of the pupil is of special importance, particularly if of the Argyll-Robertson variety. This in 90 per cent. of cases means either tabes or general paralysis of the insane. On the other hand, either of these diseases may occur with normal or only sluggish pupil reactions.

The ocular sympathetic fibre must be investigated at the same time. The result of stimulating or paralytic lesions are:

Stimulating.	*Paralysing.*
Large pupil.	Small pupil not dilating to shade.
Exophthalmos.	Enophthalmos.
Retraction of upper lid.	Ptosis.

Fifth: Motor for muscles of mastication, sensory for face, mucous membrane of nose, mouth, tongue, lips, eyes; corneal reflex depends on the nerve.

Paralysis of the fifth, seventh and eighth cranial nerves occurs as a syndrome in tumours occupying the cerebello-pontine angle.

Seventh cranial nerve supplies the muscles of facial expression; paralysis therefore causes want of expression of one side of the face, with inability to close the eye or to whistle. If the nerve is injured within the pons, the sixth nerve of the same side may be paralysed in association with it. Should the [redacted]nage occur in the Fallopian aqueduct there will be, in addition to the facial palsy, hyperacusis for loud sounds, and loss of taste on the corresponding side of the tongue owing to paralysis of the nerve to the stapedius, and to implication of the chorda tympani.

If the nerve is affected outside the sterno-mastoid foramen there will be uncomplicated facial paralysis.

Ninth, tenth, and eleventh nerves.—If these are damaged motor disturbances will result in paralysis of one side of the palate and of the vocal cord on that side; also of the sterno-mastoid and trapezius if the spinal part of the eleventh is involved. There will be some dysphagia. The palate reflex will be lost and taste affected on the posterior part of the tongue.

Twelfth nerve.—If this is paralysed there will result loss of power with muscular atrophy of one half of the tongue. We must defer considerations of the rest of the motor system to the next lecture.

December 16th, 1912.

TWO LECTURES

ON

CRANIO-CEREBRAL SURGERY.

Delivered at St. Bartholomew's Hospital,

By L. BATHE RAWLING, F.R.C.S.,

Surgeon, with Charge of Out-patients, at the Hospital.

Lecture II.

Gentlemen,—Before commencing my lecture to-day, I want to show you a very simple apparatus for taking the blood-pressure. You will remember that I spoke of blood-pressure in my last demonstration. All that is necessary is to wrap this band around the arm above the elbow and to connect up the bulb and sphygmo-manometer to these two rubber tubes, pumping in air until the radial pulse becomes imperceptible. This patient's blood-pressure is equal to 140 mm. of mercury.

Then we let out the air and remove the bandage. It is altogether simple, and a blood-pressure reading can be obtained in about half a minute. It is an American instrument, know as the "Tycos" sphygmo-manometer. I can guarantee its accuracy because I have had mine tested periodically against mercurial manometers. The normal blood-pressure is equal to about 125 to 130 mm. mercury. Blood-pressure readings are of very considerable value in estimating the degree of compression in the various head injuries and diseases.

To-day I want to deal with some points about tumours of the skull and brain. We must, I am sorry to say, take it as a definite fact that but a very small percentage of brain tumours are amenable to radical surgical treatment. The proportion has been estimated at about 5 per cent. At any rate, judging by my own experience, but few cases are capable of radical treatment, though now and again one encounters cases in which the results are quite satisfactory. I would remind you, however, that you are usually entirely ignorant of the nature of the tumour until you have seen it. Surgical treatment, therefore, is generally demanded in order that you may see the tumour and estimate whether it is removable or not.

This first slide illustrates a glioma of the brain. Gliomata form perhaps the commonest type of brain tumour, but it is only in their early stages of development that they are capable of removal, and even then it is not at all clear whether the attempt should be made or not. They are not nice tumours. One must regard these gliomata and glio-sarcomata as tumours which infiltrate and present no definite margins. They penetrate deeply into the brain substance and are usually incapable of removal. Here is a slide showing a pure sarcoma of the brain. Sarcomata are in their very early stages more or less encapsuled and capable of removal. Whether they always recur or not is rather doubtful. In the majority of cases they do, but quite early they show some encapsulation and they may be shelled out. To obtain such a result they must be attacked early and fully exposed. Obviously also it is necessary that they should be situated near the surface of the brain.

The next slide shows a sarcoma of the brain which has gone beyond the possibility of removal. Its margins are ill-defined, and any attempt at removal would have been followed by disaster.

The next picture shows a fibroma of the dura mater, and in this specimen you will see that the tumour is attached to the inner surface of the dura mater, merely indenting the brain, to which it is connected by a few strands of pia-arachnoid. If this tumour had been diagnosed and exposed it could have been removed with the greatest ease. Next I show you a slide illustrating an endothelioma of the dura mater. Endotheliomata are a fairly common type of tumour. They arise from the membranes of the brain, and, in the majority of cases, they are innocent, unless they are allowed to remain, in which case they may undergo degenerative changes. But for a long period of their existence they are well encapsuled, and, if exposed, they can be removed with comparative ease. Next I show you an illustration of two tuberculomata of the brain or meninges. They lie above the tentorium cerebelli, symmetrically disposed, one on either side of the falx cerebri and in relation to the poles of the occipital lobe. They are not nice tumours to encounter even when correctly diagnosed. They are often multiple, and, what is still worse from a surgical point of view, they are frequently associated with some degree of tuberculous meningeal infiltration. So one cannot regard tuberculous tumours as satisfactory ones with which to deal. The next is a tumour of the brain, to which I would specially direct your attention for a minute or two, because their existence is becoming more appreciated. It is a tumour of the posterior fossa, lying immediately behind the petrous bone. I show you the seventh and eighth nerves, to the sheaths of which these tumours are closely connected. By reason of the fact that they are more closely related to the eighth nerve, they are often known as acoustic tumours. They are, in their early stages, pure fibromata, but if allowed to remain they undergo degenerative changes, invading the internal auditory meatus and growing into the lateral lobe of the cerebellum. If they can be reached, these tumours are removed with considerable ease. When a patient presents the general symptoms of brain tumour — optic neuritis, headache, vomiting, etc.—and in addition is stone deaf in one or both ears, you should entertain a strong suspicion that it is a tumour of this sort, more especially if an examination of the external and middle ears reveals nothing abnormal. The next picture shows the base of the skull and

the probable position of the tumour—in close relation to the internal auditory meatus. These tumours are best exposed by cutting away a large portion of the bone of the cerebellar fossa, and at a later stage opening the dura mater and working between that membrane and the cerebellum. If you can get the tumour away before it attains any considerable size the prognosis is very fair. They are regarded as amongst the most favourable types of brain tumour. Here is another type to which I would direct your attention, a tumour of the pituitary body. Here is the pituitary body of a boy who was under my care some years ago—a huge tumour, as you can see, occupying the position of the pituitary fossa. It was of the cystic type, and we called it a cystic sarcoma. The size of the tumour can be appreciated from this picture, and the symptoms are of great interest. Presently I will direct your attention to the visual changes observed in many of these cases. The more general symptoms are of equal interest. There are two different conditions observed in connection with these pituitary tumours. There is the type in which there is hypersecretion of pituitary material, and the type in which there is hyposecretion. In the former instance the patient suffers from acromegaly or giantism. You find that these patients have big hands and feet and a big tongue, with mental lethargy, loss of hair, and other symptoms associated with that condition. In practically every case of acromegaly the pituitary body is at fault, with well-marked hollowing out of the pituitary fossa. These cases are regarded as examples of hyperpituitarism. Hyposecretion, according to Cushing and others, is characterised by excessive adiposity in the young, especially in girls, with what may be called persistent infantilism, both bodily and mental. There is a marked retardation in the development of sexual characteristics.

These points with respect to both increased and decreased secretion are still more or less *sub judice* but, so far as my experience goes, it is certain that nearly every case of acromegaly shows an enlargement of the pituitary body. We will not enter to-day into a discussion as to the treatment of these cases. You will hear more about them next year. This next picture is to show you the visual symptoms which occur in cases of pituitary enlargement. Here is a diagram showing the course of the optic fibres, with special reference to a lesion of the occipital lobes. The pituitary body lies in this angle, and, on tracing these fibres forwards, you will see that a pituitary tumour would produce blindness in these two halves of the retinal field, the patient suffering from bitemporal hemianopia. That is one of the important symptoms in pituitary enlargement, though my own experience would tell me that it is a comparatively rare symptom, less constant than acromegaly, giantism, etc. But whenever you observe these symptoms in a patient you can be fairly certain that you have to deal with a pituitary tumour. And your next course will be to take an X ray of the patient's skull, thus verifying your diagnosis. It is impossible to enter into any further detail on these points, but you can trace in this figure the course of the optic fibres, and estimate the probable position of any tumour according to the visual symptoms observed. Later on, when the pituitary tumour becomes large and compresses the optic chiasma, optic atrophy is observed, with blindness in both eyes.

Here is the picture of another tumour, an extrinsic tumour of the skull-bones. This patient had a large firm tumour originating in the temporo-parietal region. I showed her at surgical consultations and there was no doubt about the diagnosis. My colleagues were strongly averse to my carrying out any radical treatment. I have regretted ever since that I did not do so; I believe that I could have taken it away and given her a chance of cure. These extrinsic sarcomata grow from the periosteum and grow extrinsically, only invading the dura and brain at their later stages. Here is a specimen from the museum showing the intrinsic type of sarcoma of skull-bones. The whole thickness of the skull is involved and the growth has begun to infiltrate the brain. It is useless to attempt to remove a tumour of this nature. Nothing can be done unless you like to try Coley's fluid, radium, or any other such doubtful remedy.

Now let us consider the question of treatment of brain tumours in general. Here I think one may lay down definite statements. You diagnose your case first of all. There are the more general symptoms—headache, vomiting, optic neuritis and so on. And then you endeavour to localise the tumour to some special area of the brain. And here you often get into difficulties. I do not think that any surgeon is absolutely justified in going for a brain tumour until the case has been

investigated by a medical colleague ; the localising features are often deceptive, and the whole success of your operation depends on accurate localisation. If you go burrowing round for a tumour, and more especially when the localising features are deceptive, you will get into difficulty, and the mortality in such cases at once becomes high. But if the tumour be accurately localised there is a good chance of being able to expose it and possibly of removing it. All attempts at tumour removal are dependent for their result on the non-development of shock, and this is itself dependent on the control of bleeding. From the very beginning to the end of the operation it is absolutely essential that not one drop of blood should be lost more than is essential. In connection with this, please bear in mind the probable duration of the operation. Ether is the anæsthetic of choice in America, whereas in this country we are more inclined to chloroform. Anyhow, whatever the anæsthetic, one thing stands out pre-eminent in the development of shock, and that is hæmorrhage. Therefore when you start out to make an exploration of the interior of the skull, as the operation is certain to take some time for its completion it is essential in the first place that you should control the scalp bleeding. You know full well, when you turn down a scalp flap, how it bleeds, and you know how the forceps applied to the bleeding points continually slip away from the tissues. The vessels spurt freely, blood covering the field of operation, obscuring one's view and wasting valuable time. Moreover, whilst this is going on, the patient is losing strength, and this is at the very beginning of the operation. Therefore in all these cases, if you are going to expose the tumour by osteo-plastic flap, the first thing you have to do is to apply a sheet of gauze over the patient's head so as to protect the wound from infection. Then apply the scalp tourniquet, which, as I told you before, consists of an india-rubber band passing circumferentially round the head, tied almost as tightly as you can. If applied with the desired degree of pressure, all bleeding from occipital, temporal and other scalp vessels is efficiently controlled. Here are two pictures showing the method in greater detail. I have used the scalp tourniquet during prolonged operations, and, so far as my experience goes, I have never seen any ultimate remote ill-effects from its usage—there is never any sloughing of tissues, etc. Thus you are provided with plenty of time for your operative procedures without fear of hæmorrhage during the earlier stages of the operation, and there are no remote ill-effects.

Next, bear in mind that the majority of tumours exposed by operation are situated in such regions of the brain as will provide some definite localising symptoms by means of which we can know their general position. I am leaving out cerebellar tumours for the time being, having insufficient time in which to discuss them. The majority of cases of brain tumour which come under the care of the surgeon are associated with fits, or with such definite motor or other disturbances as will enable you to form a fairly accurate opinion as to the probable site of the tumour. In such instances, the tumour will probably be situated sufficiently high as to permit of the application of the tourniquet.

Next, as to the actual methods of procedure. Bear in mind that in your treatment of the tumour there are two possibilities : one is to expose the tumour to see if it is capable of removal, and the other is to remain satisfied from the very beginning with a pure decompression operation—that is to say, one which will so lessen the compression as to cure the optic neuritis, relieve the headache, etc. In this latter case you make no attempt to find the tumour. That kind of operation is obviously suited to cases in which the symptoms are so ill-defined that you are unable to localise the tumour with any degree of accuracy. We will suppose, however, that we have to deal with a tumour which is localised to the Rolandic region, the nature of the tumour and its possibility of removal being otherwise quite unknown. An important point is this. The great majority of brain tumours should be exposed. I think one can say with some certainty that if you expose a tumour and find it irremovable, and do not go any further, you do not do your patient harm. But if you find it irremovable, and yet make an attempt at its removal, the mortality at once goes up. Let us now return to a brain tumour which you are going to expose by osteo-plastic resection. This procedure was first carried out by Wagner, and his method is very similar to that utilised at the present day. First of all put on the scalp covering and tourniquet. Then three incisions are made down to the bone, in a U-shaped manner. You do not turn the scalp down, merely

displacing it in such a manner that you can see the bone along each of the three scalp incisions. At the two upper angles of the flap, with a small trephine, take away two discs of bone, then pass underneath the bone, from one trephine hole to the other and between the bone and dura mater, the Gigli director. The point of this instrument is tilted up, and by keeping close to the bone there need be but little fear of injuring the dura mater. Along the grove in the director pass the Gigli saw, apply the handles, and by gentle side to side traction, complete the section of the bone between the two trephine holes. That being done, the next thing is to cut two channels in the bone along the line of the two vertical scalp incisions with de Vilbiss forceps. Some people prefer a different method. They make two more trephine holes and again use the Gigli saw. Anyhow, the bone having been cut through along the line of the three incisions, the base of the flap remains. If you have made your incisions in such a way that they converge below, the base of the flap is readily fractured. When, however, the base is rather broad, turn the forceps slightly inwards so as to weaken the base. Then, getting two elevators beneath the flap, it is seized with the hands and broken across at its base.

That is the first stage of the operation. It is inadvisable to do more on that day. The patient may be left for seven to ten days. The flap is put back in its original position, anchored with a few sutures, a firm circular bandage applied and the patient sent back to bed. In ten days' time he is re-anæsthetised and the bone flap turned down, the dura opened and the tumour removed. I would warn you that the meningeal arteries may be troublesome, and therefore, before opening the dura mater, as you see in this illustration, it is necessary to apply ligatures to the various branches on either side of the line proposed for dural section—under-running with a small curved needle threaded with the finest silk. The area of brain exposed is proportionate to the size of the flap. Remember that we are working in the Rolandic area, and though one may be able to say that the tumour is in the immediate neighbourhood, it may either be in front or behind of the Rolandic fissure, and consequently the greater the area of brain exposed the better are your chances of finding the tumour. When exposed, you estimate whether it is advisable to go any further or not, and it is at this point that the whole success or want of success lies. The making of the osteoplastic flap is merely a mechanical procedure which anybody can do; the vital question remains as to how much further you should go and what you can do in any given case. It is in this respect that the mortality is mainly concerned. I hold strongly that unless the tumour is absolutely favourable for removal in every respect you will be very unwise to make the attempt. If removable you can proceed. In a small proportion of cases you will obtain a successful result. After removing the tumour the dura is sutured in its original position and the osteoplastic flap replaced, anchored by a few sutures. Moreover, your patient will have what one may call a sound head.

If you find that the tumour is not removable, as is so frequently the case, what will you do then? If you put the dural and bone-flaps back, you will have done your patient no good at all. One wants in such cases to carry out at any rate some method of decompression—to relieve the symptoms. Therefore you will ask me how can you decompress a patient when you have already formed an osteoplastic flap. One way is to remove the bone in the flap, leaving the dura open, and with a mere scalp flap covering that part of the brain which has been exposed. That is a bad method, because the brain which you have exposed will then herniate outwards, with subsequent and consequent spasticity and paralysis of the opposite side of the body, and perhaps the speech centre will also be involved. So although this procedure may relieve the patient's headache and optic neuritis, you will give him paralysis instead. This would not be a good surgical result. Here is a method which is exceedingly applicable and which has been carried out with considerable success. Here is a picture showing osteoplastic resection, with the dura mater sewn back into position as if you were going to replace the bone-flap also. Now, when the dura is sewn up, cut away the bone from the lower part of the bone-flap and cut away also the bone from the temporal fossa, freely and low down over the temporal fossa—below the level at which the flap was fractured. Then incise the dura mater freely—low down over the temporo-sphenoidal lobe. You will remember that I said that if you expose the temporo-sphenoidal lobe and allow it to herniate no unpleasant symptoms result; that region can herniate as much as it pleases without producing any

disastrous results, either motor or sensory. This method is known as osteo-plastic resection followed by decompression. It is one of Cushing's operations, and it seems to me, both from my own experience and from that of others, to be one of the best methods of treating those cases in which a tumour has been exposed and deemed irremovable. An important point in connection with this operation is this: it is all very well telling you to sew up the dura mater, but if you are dealing with a brain under tension you will find it not so easy of accomplishment. Having turned down the dural flap, the brain will herniate outwards, and it is often impossible, unless you carry out certain methods to reduce the protrusion sufficiently to enable you to fulfil your object. Here are some methods which will help you. First elevate the patient's head so that the blood gravitates to the body proper. Another method is lumbar puncture. If, finding that you cannot get the brain back, you lumbar puncture the patient, you will often find that the hernial protrusion will diminish sensibly. Allow the cerebro-spinal fluid to leak away slowly, then you may be able to replace the brain and sew up the dura. You can also "milk" the pia-arachnoid, though I doubt if this procedure is of much use. Another method is to puncture the lateral ventricles or even to aspirate the tumour itself, this last-named method being of great assistance if the tumour is of a cystic type. By a combination of these methods you may hope to get the brain back sufficiently to allow you to sew up the dura. Then deal with the bone. Another little thing I might tell you is this: you often find there is some troublesome bleeding from a small vessel on the surface of the brain or from the dura—a vessel that has burst. You find it is difficult to stop this bleeding by ordinary measures. First apply pressure by little hot pads wrung out in hot water at a temperature between 110° and 115° F. But this method often fails; then a useful measure for the stoppage of this bleeding is to apply muscle grafts. In nearly all these operations you have the temporal muscle exposed. Snip off a little piece, dry the bleeding point, and just place your muscle graft on that point, keep it there by the pressure of the finger for a minute or two, and you will find, in the majority of cases, that the graft adheres and stops the bleeding. It is a useful measure.

Then comes the other form of treatment. You have from the first considered it inadvisable to expose the tumour, either because the patient is not fit for radical operation or because the tumour is growing rapidly. You have at any rate made up your mind that radical measures are hopeless. You are anxious, however, to alleviate the symptoms, to cure the optic neuritis, relieve the headache, etc. If you allow the optic neuritis to progress it will end in optic atrophy, and if the patient lives sufficiently long he will have added, to his other distressing symptoms, blindness. An adequate decompression will probably cure the optic neuritis and headache. You can decompress your patient in the manner I showed you last time in two situations, either over the cerebellum or over the temporo-sphenoidal lobe. Over the cerebellum you obtain the greatest degree of decompression. And although I do not like cerebellar decompression for traumatic cases, yet it gives much better results in cases of tumour formation. If the patient has a cerebellar tumour it is obvious that a cerebellar decompression is the most likely to benefit. But it is somewhat risky. Bleeding is troublesome, respiratory troubles often occur during the operation, and the results are rather problematical. Therefore one cannot help concluding that a temporal decompression, either on the one side or on both, is more generally satisfactory. The operation of choice is the intermusculo-temporal operation. The incision starts immediately above and behind the external angular frontal process, curves backwards along the line of the temporal crest, and terminates in front of the tragus of the ear. Then incise the temporal fascia in the same general direction, thus exposing the temporal muscle. This muscle is split in the direction of its fibres and well retracted, after which the bone is trephined. The bone is here very thin, and the trephining should be carried out with great care so as to avoid injury to the dura mater. The success of the operation will depend on your technique, and, therefore, if you start off by injuring the middle meningeal arteries, the operative field becomes obscured with blood and considerable difficulty will be experienced before the bleeding is brought under control. Lift up the disc of bone and separate the dura from the bone with Horsley's dural separator. Then cut away the bone, forwards, backwards and downwards. Do not go upwards

more than you can help because of the lower Rolandic areas where the speech and other important centres are situated. Cut the bone away short of this and then incise freely the dura mater. Here the operation, so far as progress is concerned, terminates. You allow the cerebro-spinal fluid and brain to pass out and bulge through the opening, thus reducing the intra-dural pressure—the object of your operation. In order to minimise any hernial protrusion, sew up the temporal muscle over the brain, and the temporal fascia over the muscle. Then bring your scalp-flap back and sew it in position. The wound should be healed in ten days. Infection, so far as my experience goes, never occurs, more especially if you carry out those protective measures alluded to previously. The general results are exceedingly good. In this particular case the operation was carried out on the right side of the head—that is the side for choice for on the left side there is Broca's area coming fairly low down. It is best to operate on the side of the tumour, but in the absence of all localising symptoms the right side should be chosen. The degree of relief is, as I have said before, proportionate to the size of the tumour and the size of the gap which you make. Sometimes the relief endures many months and even years, but even if you relieve your patient for a few weeks only you will have brought about much benefit. In every case in which I have carried out this operation the patients have shown considerable improvement.

Here are some specimens which I want to show you illustrating some of these remarks. Here is a fibroma of the brain. Here is a tumour which might have been removed if we could have got at it. It is just sufficiently encapsuled to enable one to shell it out. Here is a sarcoma of the brain not absolutely encapsuled, but moderately so. If it had been diagnosed a month or two earlier it could have been removed. The next specimen shows a pituitary tumour. The patient, a little boy, was admitted some years ago before I knew anything about pituitary tumours. He had double optic atrophy and severe headache and vomiting. He also had some unsteadiness of gait, and he was diagnosed by the neurologist as having a cerebellar tumour. We exposed his cerebellum and found nothing. He was in the hospital several months, and, as his condition was hopeless, we got him into a home, where he died. I managed to get a post-mortem examination, and it was then that I appreciated for the first time the significance of his symptoms. It is a typical example of a pituitary tumour. Some people called it a cystic adenoma, others said it was a cystic sarcoma.

The next specimen shows you a horrible condition, a cystic sarcoma, such as no one would like to have to operate on. When the dura is opened in a case like this the brain would herniate tremendously, but if the cyst were punctured, the brain would recede and you would be able to sew up the dura mater again, after which you ought to carry out the method of decompression to which I previously alluded.

Next I show you a specimen of an acoustic tumour. You see its attachment by a narrow pedicle to the seventh and eighth nerves. I once operated on a boy who had double acoustic tumours, both of enormous size. The boy had a short thick neck and there was but little space wherein to operate. Both tumours were removed, but the boy died two or three days later from hyperpyrexia. He had been stone deaf in both ears, his discs were atrophic and his headache incessant. Microscopically the tumours were fibromata.

Here is an enlarged pituitary fossa. I mentioned acromegaly as a prominent symptom in hyperpituitarism. Giantism is another associated condition. All giants have, so it is thought, pituitary tumours. The famous giant O'Brien, whose skeleton is in the Royal College of Surgeons, had one of these tumours. They generally die comparatively young. Here is a tumour in the middle of the cerebellum, a hopeless kind of case from the surgical point of view. Here is another equally bad, an infiltrating subcortical sarcomatous tumour. Next I show you tuberculous tumours, three in number in the same brain. Here is a gumma. Do not go pushing iodide of potassium or salvarsan indefinitely in cases of tumour of the brain in the hope that it will be found gummatous. Unless the patient improves markedly within a month under anti-syphilitic treatment that mode of treatment should be abandoned. My own experience is that gummata of the brain are comparatively rare; they are certainly not amongst the commonest types of brain tumour. Consequently, it seems a great pity to waste time on the offchance that the tumour may be gummatous after all. If the patient does not improve greatly after a month's treatment he is not likely to do so at all. Added to that, Risien Russell has drawn attention to the fact that many of these gummata are quite irresponsive to anti-syphilitic treatment. They are dense fibrous masses, often well encapsuled, and no amount of potassium iodide, etc., will cure them.

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LECTURES ON NEUROLOGY.

Delivered to Post-Graduates at St. Bartholomew's Hospital.

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Lecture II.

Motor Disturbances.

Gentlemen,—This morning I want to talk about nervous disturbances which result in paralysis. When you are considering questions like that it is necessary first of all to review one or two anatomical points. If you take the nervous system as far as it is concerned with muscular power, you can divide it up into three main divisions. There is what is called the upper motor neuron, the lower motor neuron, and then the muscles themselves. So we shall have to consider the diseases which affect the upper motor neuron, next those which affect the lower neuron, and, thirdly, diseases which affect the muscles themselves, which are known as myopathies. I first want to indicate very briefly what the upper and lower motor neurons are.

The upper neuron includes the nerve-fibres which pass from the motor cortex down to the spinal cord, where they ultimately terminate in connection with the cells of the ventral horn. There are one or two points of importance in connection with that long motor neuronic path. If you look at the cortex of the brain you will see a well-marked fissure which runs down the side of the hemisphere. It is the fissure of Rolando. The area immediately in front of this fissure is the motor cortex, and on the cortex movements are represented in the following way: In the lowest part is the face area. Above that you have represented the upper extremity, then the trunk, then at the top the leg area, which also extends over to

the mesial aspect of the hemisphere. So you can have in the cortex a single lesion which affects the arm or leg or face without producing a complete hemiplegia. Thus cortical lesions are characterised by the fact that you may have either face, or arm, or leg, very much more affected than the other members of the group. As the fibres pass down from the cortex they come through the internal capsule, as it is called. If you look at this section, which is cut through the brain in the horizontal plane, you will find the internal capsule lying between the caudate nucleus, the lenticular nucleus, and the optic thalamus. The whole of the motor fibres here lie within a very small compass. The motor fibres themselves occupy the genu and the anterior third of the posterior limb of the internal capsule, and the whole lot—face, arm, leg, and trunk—are collected into that small area. Therefore a small lesion here will produce a very complete hemiplegia. Another point is that immediately behind the motor fibres in the internal capsule are the sensory fibres. A capsular lesion, therefore, is likely to be associated with hemi-anæsthesia of more or less pronounced degree, and if the lesion is large and spreads a little further back you will get involvement of the optic radiations; consequently hemianopia becomes added to the hemi-anæsthesia and the hemiplegia. Just below this level you get down to what is known as the brain stem. A lesion in the crus can be fairly easily recognised as a rule, because, as you see here, the third cranial nerve is so nearly related to the crus that if you have a lesion which destroys the crus itself, you are likely to have the third nerve implicated, and this will result in one of the forms of crossed paralysis of which there are three well-marked types: first, that which affects the eye muscles supplied by the third nerve of one side and hemiplegia on the other side, the so-called Weber's syndrome. A lesion in the pons a little lower down is apt to involve this nerve (the fifth) which you see coming through the middle peduncle of the cerebellum as well as the motor fibres. This will give the second type of crossed paralysis, namely, where the fifth nerve of one side is implicated, associated with hemiplegia of the opposite side. Lower down in the pons the seventh nerve of one side may be involved, with hemiplegia on the opposite side. That is the third type of crossed paralysis. Immediately below the pons the motor fibres pass through the medulla and decussate before passing down the spinal cord.

What are the characteristics of disease which affects the upper motor neuron? Irritation of the neuron at its commencement in the cortex produces fits. Those fits are, as a rule, of the Jacksonian type—that is to say, they may or they may not be associated with loss of consciousness; the fit always starts in a particular group of muscles and always progresses in a definite manner. After the fit there is some paresis of the affected limbs. But remember that Jacksonsian fits may occur in idiopathic epilepsy, and also remember that a lesion or tumour of the cortex may give rise, not to Jacksonian fits at all, but to ordinary, apparently idiopathic, epilepsy. I have brought to show you a patient who illustrates disease of the upper motor neuron. You notice that the paralysis is spastic in character, because injury to the upper motor neuron destroys the inhibitory power which the cortex exercises on the spinal cord, and therefore you find spinal tonus coming into play, uncontrolled by the cortex. Hence the increase in tone, which makes itself apparent by spasticity and also by alteration in the reflexes. There are three groups of reflexes which you have to consider. First of all there are the tendon-jerks. All the tendon-jerks, such as the biceps, triceps, jaw-jerk, knee-jerk, ankle-jerk, are increased. With this there may be associated clonus. The superficial reflexes, which include the abdominal, epigastric, cremasteric and plantar, are affected in various ways. The abdominal and epigastric are usually lost. The cremasteric reflex, on the other hand, usually persists unaltered. Apparently it is purely a spinal reflex. The plantar reflex becomes extensor, and that, of course, you will remember is a very important sign, because it always means interference of some kind with this upper motor neuron. The other reflexes one has to consider are the sphincters. There are various forms of incontinence which you may meet with. There is first of all the type which is so common in early hemiplegia—false incontinence or distension with overflow of the bladder. And it is important to remember that, because you frequently have to pass a catheter in the early stages after an attack of hemiplegia, and if one omits that there is a good deal of inconvenience caused by over-distension of the bladder, possibly with bad effects in the form of cystitis, coming later. So this distension over-

flow occurs as a result of shock in the early stages. Later on, if you had a fairly complete lesion of the upper motor neuron, you would get the bladder exhibiting independent reflex activity and reverting to the infantile type. As a result you have reflex incontinence, the bladder fills up to a certain amount, and when the pressure has reached a certain degree the bladder empties itself spontaneously. Thirdly, if there is a complete transverse lesion of the cord there is a flaccid paralysis of the sphincters, and the water simply dribbles out of the bladder as fast as it is secreted down the ureters. So much for the sphincters. Again, in diseases of the upper motor neuron you may have contractures, and the contractures may be so severe as to interfere with the tendon reflexes. There may be contraction of the knee-joint, so that the knee-jerks can no longer be evoked. The contracture in this upper neuron disease differs from that which occurs in a lower neuron lesion, because in the first case you have the strongest muscles going into a spastic condition and therefore overpowering the weaker. So contracture in the arm commonly assumes the flexed position, with flexion of the elbow and at the wrist. In lower neuron contracture there are the paralysed muscles stretching, and there are the antagonists, if not paralysed, shortening. For instance, in forms of infantile paralysis, involving the anterior tibial group of muscles, you have contracture with plantar flexion of the foot. This is due partly to gravity, as these muscles, being weak, the weight of the foot causes them to stretch, and, in addition, there is the unopposed action of the posterior tibial group bringing about contracture of an active kind instead of the passive variety met with in upper neuron disorders.

In diseases of the upper motor neuron *associated movements* in the paralysed limb can be demonstrated. In cases of hemiplegia you are sometimes rather bothered in making up your mind whether it is a hysterical or an organic condition. If you stretch your hemiplegic patient out on the couch, make him fold his arms, and then ask him to raise himself into the sitting posture, in cases of organic disease the paralysed leg will be lifted off the couch as the patient endeavours to raise himself. Flexion of the thigh on the pelvis takes place as an associated movement in organic hemiplegia, whereas if it is hysterical there will be no elevation of the leg at all. It is a test which is practically always successful. Another way in which you can bring out associated movements is to ask the hemiplegic to squeeze your hand hard with his sound hand. The other hand will go into flexion in a still more pronounced way—a condition of affairs which he cannot bring about voluntarily at all. And if you have him sitting on the end of a couch and make him squeeze hard with his sound hand, the paralysed leg comes up—a movement which does not occur in hysteria. You will notice that there is practically no muscular wasting present, or at all events, not more wasting than the disuse of the limb brings about. Lastly, there are no electrical changes in the muscles. We shall have to say more about electrical reactions on another occasion. *If you contrast a lower with an upper neuron lesion* you find, first of all, that the paralysis is a flaccid paralysis, because the spinal reflex arc is interfered with or destroyed. The reflexes, where they are affected at all, are abolished. And contractures take place, but they are active as opposed to paralytic contractures. Associated movements do not take place, there is marked muscular wasting, and lastly there are definite electrical changes. These electrical changes take the form of reaction of degeneration, which, briefly stated, is that the muscle loses its excitability to the faradic current, but it responds to galvanism, although in an altered way from normal. Instead of the normal brisk twitch you have a slow, worm-like, long-drawn-out contraction taking place. And not only that, but you have polar changes as well, so that whereas the cathode normally produces the biggest contraction with a current of 3 or 4 or 5 milliampères, in the fully developed reaction of degeneration the anode is the one which produces the biggest contraction with the same amount of current.

Lastly we have the myopathies, which, although there are various groups of them, all belong to the same type of disease, and pathologically are closely connected. The characteristic of them is muscular weakness. You may have the pseudo-hypertrophic form where there is enlargement of muscles, or on the other hand, a purely atrophic type. Sometimes the types are mixed. I have a couple of patients whom I will show you presently who illustrate this type of disease.

Illustrative Cases.

CASE 1.—I want to draw your attention to this

patient in order to illustrate a point I was speaking about last time we met in connection with epilepsy. The patient is æt. 49 years, and has been complaining for the last eighteen months of having attacks in which he loses himself. And he sometimes gets into a dreamy condition, in which he is not quite conscious of what he is doing. He also complains of an unpleasant taste. He says he experiences at times a very nasty oily taste, and a disagreeable smell.

His age being forty-nine, it is very important to seek for some other cause of epilepsy than the ordinary idiopathic variety.

When I was talking about the auræ of taste and smell I said they might be due to a lesion or neoplasm of the temporo-sphenoidal lobe. On examining this patient's discs one found he had definite papillœdema or optic neuritis. He has no other sign of organic disease, but I have no doubt the fits are not idiopathic in origin but have a definite cause in the form of a neoplasm. I have brought him here because the case illustrated so well the point I wanted to lay stress on in my first lecture, *i. e.* the importance of examining the optic discs and of suspecting organic disease particularly where fits occur in adult life. No defect of vision has been bothering him—in fact he says that lately it has been rather better than usual. There is nothing at present to indicate on which side the lesion is, so he will have to be kept under observation for a time.

Case 2.—This girl I show you is æt. 8 years, and the history is that she was quite well at birth, but at the age of thirteen months she had an illness of some kind. We do not know what it was. She appeared to be perfectly normal up to that age, but from that date her lower limbs were affected, and she has never walked at all properly. At the same time she is quite intelligent. She has an extraordinary spastic gait. Her arms, however, are not affected; she can knit and sew, and also read and write like a normal child of that age. But she has this very disabling condition of her legs. I expect there is no doubt she had encephalitis in infancy, that is to say a condition of the cerebral hemisphere quite analogous to acute poliomyelitis, a disease which of course most commonly affects the spinal cord. But whatever it was it has left her in this position. Her condition corresponds closely with the disease known as Little's disease, except that Little's disease occurs, as a rule, from birth. We have to decide what we shall do with this child. Her defect in walking is a very great disability. One wants to know whether one can improve the condition of her legs. It is possible to improve them, only there is a certain risk about the procedure which is necessary in order to bring this about. Spasticity of the legs depends on the integrity of the reflex arc, the afferent and efferent paths from the lower limb. If you take a frog and hang it up and cut its posterior nerve-roots on one side, the leg of which those roots are cut will hang down straight, whilst the other will be flexed, and have some tone in it. You can apply this lesson learnt from the frog to the human being. You can divide a certain number of posterior roots. But you must not divide them all, or else you will change your spastic patient into a tabetic, which would be to make matters worse. You can divide a certain number of roots but not enough to produce ataxy, but enough to do away with the spasticity of the legs and the spasm. And that has been done in a good many cases, not so much in this country as in Germany. Some of the results have been extraordinarily good. Children who were even more spastic than this have been able, as a result of this operation, to walk freely and step out well. As a rule three posterior roots are cut on each side—the third and fifth lumbar and the second sacral. You do away with the spasm in that way, and it is surprising that you produce practically no alteration in sensation at all. That is because the overlap of the sensory fibres in the roots of the lumbar and sacral plexus is so large that you can cut practically half of them without causing loss of sensation at all. In this child the question arises whether her disability is so great as to make it worth while to run the certain amount of risk which always follows an operation inside the dura. That is to say whether the gain which you might expect from it would compensate for or justify the operation. I certainly think it is justifiable, and I think I shall recommend her mother to have it done. Because if it is done carefully the ill-effects are *nil*. On the other hand, she stands to gain a good deal. The case illustrates upper neuron paralysis, and it is an example of the type of case which can be cured by section of these posterior roots on both sides. Sepsis is the only danger, and sepsis here means general meningitis.

Cases of this kind can be improved by massage and exercises, but in this case these have been tried and operation alone will probably cause further improvement.

The next two cases that I am going to show you also exhibit spastic paraplegia, but from quite different causes.

CASE 3.—This patient has, as you see, a very spastic gait, but he is also ataxic, and in consequence hardly able to walk alone. His history extends back for ten years, when he first complained of unsteadiness in walking, and diplopia. Since then his symptoms have varied from time to time, and have included precipitant micturition and paræsthesiæ of the upper and lower extremities.

If you examine him you will find that the special senses are normal. His pupil reactions are normal; there is very slight unsteadiness of ocular movements; it does not really amount to nystagmus, and as far as his other cranial nerves go, I think they are normal. His discs are pale. There is practically no tremor of the hands or arms, but there is a little ataxy of the arms—some wandering at the end of the effort. There is some loss of deep sensation in his right hand, which has resulted in astereognosis. He has very slight tactile loss. You saw that the legs are spastic, and there is a very brisk knee-jerk. There is an extensor response in the great toe, and ankle clonus on the left side. The diagnosis in this case is certainly disseminate sclerosis, but it is interesting to note how much it differs from the classical picture of that disease. Nearly all the cardinal points are absent in this man. There is no nystagmus or speech defect—no definite intention tremor, and though the discs are pale there is no evidence of optic atrophy. But the history is quite typical, and the case represents a fairly common type of the disease. It is important to remember that it is the exception, rather than the rule, to find a typical text-book case of disseminated sclerosis.

CASE 4.—Here is another man with a very spastic gait. I will tell you his history. He is æt. 42 years, and he is a soldier. Fourteen years ago he had gonorrhœa at any rate, whatever else he had. And eight years ago he had transient weakness of his legs; it progressed, and there was some difficulty with the water. His condition went on getting much worse until about a year ago. A year ago the condition had progressed to almost what you see now. There had been steadily increasing paraplegia. The arms were not affected at all, and he never had any trouble with his sight. There was some numbness of the legs, but objectively there is hardly any sensory loss in the legs; so clinically it appears to be a pure spastic paraplegia. And you have to find out what is the cause of that. When you have a spastic paraplegia of this kind, progressive in character, going on seven or eight years without any definite sensory loss, you can at once exclude pressure on the cord from tumour or from caries, or whatever it may be, because by this time no doubt had such been the case you would have had definite sensory loss accompanying the spastic paralysis. So we have to consider what the possibilities are for producing a condition of this kind. There are various things which you naturally think of, and one of them is disseminated sclerosis. The second is progressive muscular atrophy (amyotrophic lateral sclerosis), because progressive muscular atrophy is practically always associated with degeneration of the upper neuron as well. In these cases lateral sclerosis may be present for a considerable time before muscular wasting begins. There is no evidence of muscular atrophy in this case, and as he has had this condition eight years the probability is that it is not a case of amyotrophic lateral sclerosis. There is no evidence that it is disseminated sclerosis, and as the disease has existed ten years it is unlikely to be so. Disseminated sclerosis would hardly exist purely as a spastic case for so long without showing some other manifestation, such as nystagmus, diplopia or ataxy. And that brings us to the third alternative, and what I consider to be the most probable condition present, namely that we have here a specific affection of the spinal cord. When you come to consider syphilis of the spinal cord you find that it may exist in several different forms. The commonest form in which it occurs is meningo-myelitis, that is to say, a syphilitic condition of the meninges with resulting myelitis from vascular disease of the cord. That condition is usually a slowly progressive one, and it varies very much in regard to the amount of loss of sensation which is produced. There may be very marked sensory loss, but in many cases you do not find a very great deal. It is a condition which affects the motor tracts far more than it involves the sensory, but

there may be thrombosis taking place at any time, causing transverse myelitis, with its characteristic motor and sensory disturbances. But as a rule there is a slowly progressive spastic condition of the legs, with very little sensory loss attaching to it. Another type is what is known as syphilitic lateral sclerosis, where there appears to be simply a degenerative condition of the pyramidal tracts analogous to the degeneration of the posterior columns which takes place in tabes. Instead of the syphilitic poison affecting the posterior columns it affects the lateral tracts, and it is not very common. I think probably that is not the condition in this man. A curious point about that type of syphilitic disease is that, as a rule, with the increased reflexes there is a hypotonic condition of muscles. The other syphilitic types we do not need to consider here in detail, but one of them is gumma of the cord, which produces an acute onset of symptoms and disturbance of the sensory paths as well as the spastic condition. The fourth is chronic poliomyelitis, a chronic muscular atrophy produced by syphilis. The fifth is gummatous invasion of the posterior and anterior roots. I think the diagnosis in this case is chronic meningo-myelitis, in which the sensory paths are very slightly affected.

CASE 5.—The next case illustrates a type of lower neuron disease. The patient is æt. 28 years. He first noticed the condition affecting his fingers eighteen months ago. At that time they began to become weak, and since then there has been progressive weakness of the hands and forearms, and nothing else at all. If you examine the patient you will see he has wrist-drop, that there is a tendency to flexion on the part of the fingers, and that there is a good deal of vaso-motor disturbance about the hands, which are definitely blue. You will see that the disease not only affects the extensors, but that the flexors of the forearm are also affected, particularly on the left side. The grasp of the left hand is very weak; the small hand muscles are very weak. The inter-ossei are wasted. I do not think there is much fibrillation of muscles here, but when there is muscular wasting of that sort you look out for fibrillation. The disease developed rather rapidly, and consequently the electrical reactions show definite reaction of degeneration. There is no sensory loss, and the examination of the rest of the nervous system is negative. It is a case of chronic anterior poliomyelitis, or progressive muscular atrophy, which is running rather a rapid course. The patient has had syphilis, but the Wassermann reactions in the blood and cerebro-spinal fluid are indefinite. It may be that syphilis is the cause of the condition in this case, but whatever the cause, it does not seem to make much difference in the prognosis, which I am afraid is very unfavourable.

CASES 6 and 7.—These two children are examples of myopathy. You see there is considerable development of the calf muscles—in fact they look much too good. Notice the pseudo-hypertrophy of the spinati. The glutei are also enlarged and there is some lordosis, but not very much. The pectoral muscles are defective in the sternal part, and that is nearly always absent in these cases. The latissimus dorsi is very defective. He shows the typical method of rising from the floor by climbing up the legs owing to weakness of the glutei and back muscles. Next I show you his elder brother. There is another brother between these two who is all right. This brother has the characteristic "cake-walk" of a pseudo-hypertrophic case. There is tremendous lordosis. He has very big calves, and the feeling of them is quite characteristic; they are extraordinarily firm. You can practically diagnose the myopathic muscular condition by the feel of the muscle. There is not so much hypertrophy of his spinati as there was in the other case, but there is winging of the scapula, and there is considerable atrophy of the upper arm. This is a case where muscular atrophy and pseudo-hypertrophy exist together. It is a mixed type of the disease. Later on contractions develop and the patients become bed-ridden. As a rule they die from pulmonary complications. It is a family disease which appears almost entirely in the male members, but is transmitted by the females, in which it resemble another familial disease, namely hæmophilia.

December 23rd, 1912.

Roentgenoscopy of Adhesions Binding Down the Colon.—Pers calls attention to the instructive evidence afforded by attempts to push the colon around as the patient is being examined in front of the screen. The normal colon can be readily pushed around, but when bound down by adhesions this cannot be done, as is readily seen in the shadow on the screen.—*Journ. A.M.A.*, vol. lix, No. 22.

THE PHYSIOLOGY OF BATHING.

By R. FORTESCUE FOX, M.D., F.R.Met. Soc.,
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ALTHOUGH the use of baths, hygienic and medicinal, is of untold antiquity, the accurate knowledge of their action is new, and we are only now beginning to understand the *rationale* of bathing, both for the preservation of health and for the prevention and cure of disease. Erroneous ideas and faulty practices have too often prevailed regarding baths. They have been given, especially in schools, too often in a haphazard, or, still worse, in a routine fashion, and sometimes with injurious effect. Heat and cold are edged tools, and cannot be used by rule of thumb, especially for the young. It is not too much to say that the time has come for the methods and details of school and public baths to be founded upon physiological knowledge, under the direction of competent medical authority.

It behoves us first of all to understand something of the functions of the skin, the great nervous and circulatory organ upon which, and through which, baths operate.

Man is by nature the least protected of the warm-blooded animals, and must therefore be the most sensitive to the effects of temperature, as well as to other external conditions. Nevertheless he is, without fur or feathers, nearly as warm as the birds. Moreover, he maintains his existence over a large part of the habitable globe in an atmosphere many degrees colder than his body, and often constantly varying in temperature. Man's unprotected and sensitive body must preserve its relatively high temperature at an even level,† for any considerable variation is incompatible with life. It is also necessary that the body, without dangerous cooling, should be kept in constant and effective relation with many external conditions, including the various forms of energy permeating the world. Upon what has been called the "reaction" or response of the individual to his environment and to these energies, the integrity of his functions, both physical and mental, depend.

The skin is to be regarded as the great heat regulator of the body. The mechanism of heat regulation is essentially a system of blood-vessels covering the entire body, which can be opened or closed from moment to moment, either partially or simultaneously over the whole surface. The network of the circulation in the skin is said to be so capacious that it could contain from one half to two thirds of the total circulating blood. The more active this surface circulation, and the more blood reaching the skin, the higher is its temperature and the greater the loss of bodily heat. When, on the other hand, the circulation in the skin is contracted, its temperature falls and the loss of heat is diminished. The sweat-glands, which, like the blood-vessels, are under the immediate control of the nervous centres, also form a part of the mechanism for the regulation of heat, any increase of perspiration causing an increased loss of heat by evaporation, and *vice-versâ*.

The skin may also be described as a nervous organ for relationship with the external world, inasmuch as it contains an expansion of the nervous system spread out over the body, which may be compared in sensitiveness to a photographic plate. This nervous surface organ is constantly engaged in receiving and transmitting to the centres in the brain and spinal cord an infinite number of impressions from the external world. It is true that these afferent impulses do not for the most part affect the conscious brain. We have reason to believe that in mankind the surface of the body is more sensitive and takes a more important part in physical and mental processes than in any other animal—I lay stress upon the relation of the skin to our mental activities. It should be remembered that it is developed from the same embryonic tissue as that from which the nervous centres are derived. It is probable that in the process of evolution the functioning of the skin and nervous centres are intimately related and proceed *pari passu*.

Such is the organ upon which, and through which, baths exert their various actions. The action of baths is two-fold; in the first place a direct or immediate action, which is limited to the tissues of the skin, and secondly an indirect or mediate action, which is transmitted through the skin and exerted upon all the organs and tissues.

* Delivered at the International Conference on School and Public Baths.

† Man "of all animals has the most constant temperature under extreme differences of external heat and cold." —Pembrey, in Schæfer's 'Physiology.'

It must be frankly admitted that at present we cannot fully explain some of the effects produced by baths, both in health and in disease. It is scarcely surprising, considering the complexity of the problem that they present. On the one hand we have in the bath many different influences operating, the temperature and other physical qualities of the water, its chemical constituents, and the influence of pressure and mechanical stimulation. On the other hand we have the human body under varying conditions, and with marked individual characters. In the power of reaction to outward conditions no two persons are alike, even in health. The time of day at which the bath is taken, the relation to meals and to exercise, and to states of fatigue, all modify the result. In conditions of ill-health all such differences become more marked, and individual characters are more strikingly shown.

We know that in a state of health the temperature of the blood is maintained at or about 98° F. (37° C.). This figure represents the balance struck by the production of heat within the body and its constant loss to the colder medium in which we live. Bodily heat is produced by oxidation or slow combustion in the living tissues, particularly in the muscles, the liver and other glands. During digestion and muscular exercise the production of heat is greatly increased. The amount of heat produced may be gauged from the fact that a healthy man has been found experimentally to lose every day about 2½ million units of heat (calories). This amount would be sufficient to raise from freezing to boiling-point 55 pounds (25 kilogrammes) of water. For our present purpose it is important to observe that in man nine tenths of this amount passes away by the skin—by radiation, conduction and evaporation. The low power of conductivity for heat belonging to the skin, and the presence of fatty tissues under the skin, prevent our rapidly growing cold, because these tissues interpose between the vital parts and the external world a non-conducting barrier. Other things being equal, the loss of heat is less in adults than in children, in large than in small animals. The ratio of surface to bulk diminishes as the size of the animal increases. In whales and other large animals living in the Arctic Seas the loss of heat is minimised, and their high temperatures maintained, partly by their great bulk, and partly by the thickness of their non-conducting skin and blubber. In man the average temperature of the skin is considerably warmer than in these animals, and approximates to 93° F. (34° C.), that is to say, from five to eight degrees only lower than that of the blood. We are protected by a cool integument against too rapid a loss of heat. Clothing, of course, further checks the loss of heat by keeping the body surrounded by a zone of warm air. The temperature of the included air in ordinary clothing is about 89° F. (31·5° C.).

It follows that when the body is exposed to air at or about 89° F. the normal values of heat production and loss are unaffected. Let the atmospheric medium be a little warmer and the loss and production of heat are diminished, or a little cooler and they are increased. At a certain point the normal exchange is undisturbed. This point of temperature, the "point of thermal indifference for air," is fixed at from 85° to 89° F. We know that in some tropical countries men spend their lives in an atmosphere approaching these temperatures. Their bodies are in a state of thermal equilibrium, and the delicate mechanism for the regulation of heat production and loss, upon which we depend for our existence in colder climates, is for them more or less superfluous. The reaction of the body to cold has perhaps never been fully developed, at all events it is not, as with us, constantly in operation, and hence cold applications should be used with caution.

Water conducts heat more freely than air. It has been found that in a bath about the temperature of the skin (93° F.) heat production and loss are unaffected. The point of thermal indifference for water is therefore fixed at 93° F. (34° C.). In considering the effect of baths, both in health and in disease, it should be borne in mind that baths at or about this temperature have no thermal result upon the body, whilst all other baths, whether hotter or colder, always affect the bodily heat. There are many natural waters employed for baths at such temperatures, like those of Leukerbad in Switzerland and Kawanaka in Japan. It is an ancient custom for persons to stay many hours, and sometimes for whole days or even weeks, in these indifferent or athermal baths. No temperature effects are experienced. It follows that when it is desirable for purposes of cleanliness or otherwise that baths should be prolonged or

frequently repeated they ought to be at or near the point of thermal indifference (93° F.), otherwise they would add to, or diminish, the bodily heat in an undesired and possibly in an injurious manner.

The immediate action of sweet water baths (excepting those of an athermal or indifferent temperature) is to convey heat to the body or to abstract heat from it, that is to say they act chiefly as a vehicle of heat. The moisture of a bath may be considered as a sedative influence, combined with the stimulating influence of heat or cold. Moisture softens and relaxes the skin, separates the dead epithelial scales, and causes the deeper cells to swell by the absorption of water.

From the point of view of the human body heat may be regarded as positive when it is above and negative when it is below the temperature of the skin. The physiological effects of baths, positive or negative, are of the same general character whatever the form of bath may be. Immersion baths of water, of hot air or of vapour, douche baths and local baths, and hot or cold packs, produce the same thermal effects, with many modifications, especially when changes of temperature are introduced.

Considering first the effect of positive heat, the immediate impression of heat upon the nerves of the skin is stimulating to the nervous centres and to the circulation. Warm or hot baths cause an expansion of the superficial arteries; blood is attracted to the surface of the body, and the skin becomes flushed and warm in the bath. Hot baths in this manner rapidly raise the temperature of the circulating blood, and at the same time prevent the normal surface loss of heat. If the bath is continued heat accumulates more and more in the body. The first stimulating effect gives place to a secondary febrile condition, and in a bath at 104° F. the temperature of the blood may rise as much as five degrees in half-an-hour. The circulation is quickened and excited, the muscles are relaxed, perspiration sets in, and a state of artificial fever is in fact produced. A condition of enervation and general debility follows, proportional to the temperature and duration of the bath. Persons who habitually take baths of this kind often suffer from a permanent loss of tone of the nervous centres and of the heart. It is said that the fondness of the Romans for warm baths was one of the causes which contributed to the decline of the Roman power. The more exact knowledge that we have now obtained of the gradual and insidious effects of long-continued heat upon the body should make us very watchful as to its employment, especially for young persons. What is called "thermal fever" and "heat stroke" are often caused in hot climates by exposure of the body to moist heat, by which the action of the skin is checked and heat accumulates in the body. This dangerous condition, which is often fatal, is an extreme form of what is produced by prolonged hot baths.

Turning now to negative heat, that is to say to cool and cold baths, the immediate effect of such applications is a contraction of the surface blood-vessels proportional to the degree of cold. This is a reflex action, protecting the body from dangerous loss of heat. The skin becomes pale from diminished blood-supply, and the circulation in the internal organs is correspondingly increased. The action of the heart becomes slower and stronger. In a short time what is known as the "reaction to cold" sets in. This reaction of the living body to cold is one of the cardinal facts of physiology. Upon it is founded the whole action of baths. The power of reaction may be feeble and inadequate, or strong and adequate. It varies with age and condition and individual character, and in the same person at different times. The signs of reaction are easy to read, and its quality can be readily measured and recorded. It has been shown that the impression of cold on the skin gives rise through the nervous system to a great increase of heat production in the body, brought about principally by the combustion of fats. Presently, with the increasing generation of heat, the first protective contraction of the surface circulation gives place to relaxation; the blood flushes the surface, and a sensation of warmth pervades the body. It is important to remember that whereas in hot baths the loss of heat is hindered or prevented, in cold or cool baths it is enormously increased. Liebermeister has shown that in a bath at 20° C., of fifteen to twenty minutes' duration, the loss of heat may be increased five-fold. Such a loss is for many persons more than can be made good. It is only when the powers of reaction are normal and metabolism active that nature can respond with a corresponding increase of heat production.

Cold may be regarded as a powerful stimulant to the circulation and to the nerve-centres, providing that these are sufficiently vigorous to withstand the shock and to reinforce the failing bodily heat by an immediate increase of combustion in the tissues.

The increased vigour of the circulation in the internal organs no doubt accounts for the muscular energy and mental refreshment experienced by healthy persons after a brief cold bath. An interesting observation with reference to such baths (duration 2½ minutes) has been made by Ignatowski. He has shown that in a healthy subject during the first minute's immersion 44 calories were lost, but in the second minute and a half, the surface temperature having fallen, the loss of heat was not quite half as much (21 calories). Meanwhile the heat production during the bath rose to fourteen times the normal. Notwithstanding these abrupt changes of heat production and loss, the general body temperature was unaffected. It is important to remember that although the temperature of the blood remains unaltered by this vigorous reaction to cold, the processes of tissue change and the arterial circulation are powerfully stimulated.

When the application of negative temperature is continued beyond a certain point a very different sequence of events takes place. The striking reaction which cold induces in warm-blooded animals shows it to be a strong stimulant, within certain limits. In excessive doses it is an equally powerful and even fatal depressant. When the cold bath is too greatly prolonged, the production of heat fails to keep pace with its loss, and the temperature of the body rapidly falls. The surface vessels, particularly the veins, become dilated and paralytic, a condition indicated by blueness of the surface. Blood accumulates in the veins, the circulation fails, and chilliness gradually passes into algidity and collapse. Deaths from exposure to atmospheric cold are brought about in the same manner, and analogous effects are seen locally in the phenomena of frost-bite.

It is a noteworthy and suggestive fact that the human body is less tolerant of continued heat or continued cold than of abrupt changes of temperature. When the surface circulation is well opened by previous hot applications, violent and extreme changes are surprisingly well borne. The northern nations have from a remote period used "sweating baths," in which the bather is exposed first to heat and then to cold. The Russian comes out of his "sweating-chamber" at 125 or 130° F. with a red, burning skin, bathed in perspiration, and rolls in the snow or in ice-cold water with impunity. In the same manner the Japanese at Kusatzu practise cold affusion immediately after an immersion bath at 120° F. On physiological grounds we may believe that such more or less active gymnastics of the skin is well adapted for dwellers in northern latitudes. It may be expressed by the formula, "*minus* after *plus*."

It remains to consider what practical conclusions as to the hygienic use of baths can be deduced from the foregoing data. Here, as elsewhere, it will be found that science confirms the result of experience. It is plain that both heat and cold have a powerful effect upon the human body. Both of them may be stimulant, sedative, debilitating, depressant, and finally destructive to organised life, according to dose and according to duration.

Baths may conveniently be classified as under, with reference to the temperature of the body:

Minus baths (negative heat)	Very cold, 32°–60° F. (0°–15° C.).
	Cold, 60°–75° F. (15°–24° C.).
	Cool, 75°–90° F. (24°–32° C.).
Plus baths (positive heat)	Warm, 90°–98° F. (32°–37° C.) (subthermal baths).
	Hot, 98°–105° F. (37°–40° C.). (thermal baths).
	Very hot, 105°–120° F. (41°–49° C.) (hyperthermal baths).

Conclusions.

(1) Cold immersion baths should be used only by persons with active circulations and a normal "reaction to cold." The degree of cold and the duration of the bath should be proportional to the vigour of the reaction. Reaction is increased and promoted when the bather is warm before entering the bath, and when the cold is rapidly applied.

(2) A cold bath, whether in sweet or salt water (sea baths), should never be continued when the termination of reaction is indicated by the sensation of warmth giving place to what is known as the "second chill." Movement of the air (wind)

greatly increases the loss of heat, and hence sea baths should only be used with caution by delicate persons and children on windy days.

(3) The brief cold "morning tub" is a good mental and physical tonic for young and healthy subjects, in winter as well as in summer. Its educative effect is seen in the cultivation of will-power. Children and others with feeble reaction should not in winter time use it below 65° or 70° F. (the temperature of "cold water" in summer), and never when feeling chilly (Dr. Duke, of Rugby). Since muscular work and digestion both increase the production of bodily heat, a cold bath may be taken with the best advantage by delicate persons immediately after brief exercise or a short time after a light meal. Many otherwise healthy individuals have a defective reaction to cold, which, however, is capable of education. It is generally defective in young children and old persons. It follows from the above that only the robust should take a bath below 65° or 70° F. without previous application of heat.

(4) Cold after heat (minus after plus) is the form of bath most applicable for all ages in cold and temperate climates. It is especially suitable for young children.* At both extremities of life loss of bodily heat is badly borne. The degree and duration both of cold and heat should be adjusted to the vigour of the individual. In school life the ideal daily bath is a brief shower, first hot and then cold, and taken when warm after vigorous exercise.

(5) Cool and cold baths when systematically employed educate the natural reaction to cold, and greatly increase the resistance of the body to fluctuations of external temperature. The common and often serious ailment known as "chill" is a result of failure of this natural reaction, from which follows diminished resistance of the tissues to the invasion of micro-organisms. The many forms of illness in cold and temperate climates depending upon "chill" as a necessary first stage would be to a large extent prevented if a healthy reaction to cold was produced in the community at large by the habitual use of appropriate baths.

* "In early infancy the bath should be at 100° F.; at six months a morning bath at 80° F. with a rather cooler sponging after; at ten months cold sponging may be used after warmth; and at four to five years a brief cold bath" (Mrs. Langdon Hewer in 'Our Baby').

(6) Cold and cool baths are not adapted for hot climates because the reaction to cold has never been fully developed in these climates, and chill is, therefore, more readily produced. It is for this reason that cold baths are dangerous in the tropics.

(7) Warm and hot baths are at first stimulating but very easily become depressing. In many, even in young persons, the action of the heart is quickly affected, and dangerous weakness may supervene several hours after leaving the bath. In robust persons a brief hot bath after strong muscular exertion promotes the removal of waste products and so prevents stiffness. In the same manner warm and hot baths often remove physical fatigue. Very brief hyperthermal baths are of all forms of bath the most stimulating. Baths above the point of thermal indifference (93° F.) should never be long contiuned, and may often with advantage be followed by an application of cold (*minus* after *plus*).

(8) When prolonged beyond a brief period, hot baths temporarily lower the tone of the circulation, the muscles, and nerve centres. When habitually used they produce a permanent condition of mental and physical debility. The habit of taking frequent and prolonged hot baths is especially injurious to young persons, and should be strongly discouraged.

(9) Brief hot baths are well adapted for use in hot countries. They promote the surface circulation and so increase the loss of heat, which otherwise tends to accumulate within the body.

(10) Baths at an "indifferent temperature," at or about 93° F., do not affect the production and loss of heat, and are suitable for cleansing purposes as well as for baths of considerable duration.

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SOME POINTS IN CONNECTION WITH URINARY TUBERCULOSIS.

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AT a recent congress of the German Urological Association, held in Vienna, one of the subjects for discussion was, "The End-results of Nephrectomy for Renal Tuberculosis." A large amount of material was brought forward, and it is a significant fact—when we compare the divergent opinions held in our own country—that the verdict in favour of operation was a unanimous one. This verdict is strongly upheld by all surgeons who have made a special study of urinary surgery. In order to form an impartial opinion on so important a

question, let us compare briefly the end-results of cases treated by non-operative measures with those treated by operation.

(1) *What are the end-results of cases of renal tuberculosis treated without operation?*—All the available literature on the subject has been collected by Wildbolz (1), who adds a valuable contribution of his own. Thus Caspar divided his unoperated cases into two groups: Those who were inoperable at the time of examination, and those who were operable but refused operation. The former all succumbed within a very few years. The latter group comprised 38 patients; in no single case did cure result; 10 died within three years, 3 others lived four, five and eight years respectively. The rest, at the best, showed no improvement at the time of writing. Of 32 non-operated cases observed by Blum, during a period of eight years, the majority succumbed within three years; only 4 of these cases, however, could be considered operable when they first presented themselves for treatment. Ekehorn's statistics show 29 cases of urinary tuberculosis (all, save one, were primarily renal) treated by non-operative means. Of these, 15 had already died at the time of writing, 8 were still alive (the duration of their respective illnesses being five to ten years). Of these 8, 4 remained *in statu quo*. In the other four, all subjective symptoms had disappeared, but in only two of them was examination of the urine possible; both these still had albuminuria, though no tubercle bacilli were found. The remaining six cases had drifted away and had been operated upon elsewhere.

Wildbolz collected 316 cases treated by non-operative measures by various Swiss doctors. These included bilateral and unilateral renal cases and cases of genito-urinary tuberculosis, the latter doubtless accounting for the long duration of some of the cases. Of these, 218 (*i. e.* nearly 70 per cent.) were already dead.

99 (31·3 per cent.)	died during the	1st–2nd	years of illness.
86 (27·2 ,,)	,, ,,	3rd–5th	,, ,,
20 (6·3 ,,)	,, ,,	6th–10th	,, ,,
5 (2·5 ,,)	,, ,,	14th–15th	,, ,,
3 (--)	,, ,,	20th–30th	,, ,,

Most of these patients died as the direct result of their disease (marasmus, uræmia, general tuberculosis and phthisis); of the remaining five cases no details were available.

Only 98 (30 per cent.) were alive at the time of the inquiry; of these, 68 still had active disease, 30 had no subjective symptoms (but no urinalysis was made). The duration of the disease in these 98 cases was as follows: In 21, 1 to 2 years; in 38, 3 to 5 years; in 18, 6 to 10 years; in 12, 10 and more years.

Wildbolz himself had treated 64 patients during the last ten years by non-operative means: of these 34 had died—all, save two, from tuberculosis: 11 lived 1 to 2 years; 17 lived 3 to 5 years; 6 lived 5 to 9 years.

Of those still living, the duration of illness had been: 1 to 2 years in 3 cases; 2 to 5 years in 8 cases; 5 to 10 years in 12 cases; 10 or more years in 7 cases.

Or to regard these 64 cases from another point of view, of 29 who had bilateral renal tuberculosis, 22 (76 per cent.) had died, 7 were still alive; of 29 who had unilateral renal tuberculosis, 10 (34 per cent.) had died and 19 were still alive. (Six, of whom two had died, were unclassified.)

Of the 19 unilateral renal cases surviving, 18 still had active disease. One appears to have been cured by tuberculin. At the present time (five years from the beginning of his illness) he passes clear urine, free from pus and albumen, and cystoscopy shows an efflux coming from each kidney.

Hottinger reported 127 cases of renal tuberculosis, treated by conservative methods: 76 per cent. had already died of tuberculosis; of the 15 survivors, 14 still had active tuberculosis; 1 only was an apparent cure.

To sum up these results: Out of over 600 patients treated by non-operative means, there are only 6 cases in which cure is claimed. Four of these are reported by Ekehorn, but in only two was the urine examined; these two still had albuminuria, though tubercle bacilli were not found. In Wildbolz's case only five years had elapsed from the beginning of his illness. Latent periods even as long as this are a well-recognised feature of renal tuberculosis; so we cannot yet look upon this case as cured. There remains one case reported by Hottinger, but no details are available.

We see therefore that 70–80 per cent. of cases treated by conservative measures die within five years. The remainder linger on for varying periods, some with intervals of remission up to

five years, others with continuous subjective symptoms, all with progressive disease.

(2) *What are the results of renal tuberculosis treated by nephrectomy?*—Israel records the results of 1023 cases (170 of which were operated upon by himself).

The immediate mortality (*i. e.* within six months of operation) = 12·9 per cent.

The remote mortality (the majority of these deaths occurred within two years of operation)= 10–15 per cent. Total mortality about 25 per cent. The causes of death were:

(1) *Pulmonary tuberculosis* 45 *per cent*, (at least 70 per cent. of these patients had pulmonary tuberculosis before operation).

(2) *Disease of the second kidney* 35 *per cent.* Of these 35 per cent., in 69·5 per cent. of cases the disease was tuberculous (in most of these the second kidney was already tuberculous at the time of operation), in 30·5 per cent. non-tuberculous.

(3) *Acute miliary tuberculosis* 14 *per cent.*

Let us now consider the condition of the survivors. Of these, 63·8 per cent. are cured, if we take as our standard absence of the tubercle bacillus in the urine. Most of those who still have tubercle bacilli present have no frequency of micturition and have put on weight since operation. In many cases the bacilli are present without pyuria, without pain, and with no demonstrable disease of the bladder or remaining kidney. This has been observed up to seventeen years after nephrectomy, and Israel looks upon such patients as analogous to "typhoid carriers." (That tubercle bacilli can be eliminated in the urine of tuberculous patients in the complete absence of urinary tuberculosis was shown by Phillipowicz [2]. Observations of Pousson [2] and Jousset [4] also support such a contention.) Cystoscopy showed that healing of the bladder had occurred in 43·5 per cent.; thus the tubercle bacillus disappears more often than the affection of the bladder mucous membrane—many of the later cystoscopic changes being no longer tuberculous.

Wildbolz reported 139 nephrectomies with an operation mortality of 2·8 per cent. Subsequent deaths numbered 20 (*i. e.* 15 per cent). These occurred as follows: 13 in the first year after operation; 4 in the second year after operation; 2 in the third year after operation; 1 in the fourth year after operation.

Cause of death.—Marasmus 3, phthisis 9 (present before operation in every case), tuberculous meningitis 4, miliary tubercle 2, peritoneal tubercle 1, pneumonia 1. (Two probably had tuberculosis of their remaining kidney at the time of death.)

Up to the end of 1910 Wildbolz had performed 125 nephrectomies. At the time of writing 102 of these patients were still alive, their condition being as follows:

Cured, 66 (no symptoms, urine normal and free from tubercle bacilli). Ten more can probably be reckoned as cures; no tubercle bacilli are to be found in the urine, though in five a trace of albumen remains, and in the other five a few pus-cells are still obtained after centrifugalisation.

Unhealed, 26.—Of these, 10 are still improving, and as two years have not yet elapsed since operation, will probably be cured. Three have chronic cystitis, non-tuberculous; 13 are still tuberculous, but all are able to follow their employment save 4; 2 of these probably have tuberculosis of their remaining kidney.

Zuckerkandl reports 104 nephrectomies. There were 20 deaths in all, most of these occurring during the first year following operation. Improved methods of diagnosis have diminished the mortality to only one in the last 50 cases. Of the 20 deaths, 7 occurred during the first four weeks; other 8 during the first year after operation; 3 died in the second year, 1 in the fifth, and 1 in the sixth year. Nearly half of the deaths which occurred during the first year were due to some other tuberculous lesion (*e. g.* phthisis) which was present at the time of operation.

Of the 84 survivors, 45 (*i. e.* 53 per cent.) are completely cured. (No symptoms, urine clear and free from tubercle bacilli, patient doing full work.) The rest are "improved"; many of them had ulceration of the bladder or genital tuberculosis at the time of operation. Most of them appear quite well, but their urine is not yet normal.

Asakura had performed 70 nephrectomies with an immediate mortality of 4 (*i. e.* 5·7 per cent.), and remote mortality of 5 (7·1 per cent.). There were no renal deaths. Of those surviving, 36 (or 51 per cent.) were cured (no symptoms, capable of work, urine clear and no tubercle bacilli), and 25 (*i. e.* 35·7 per cent.) improved. The latter were not yet capable of full work, and were still under treatment for either the bladder or nephrectomy wound.

André's results showed a total of 57 nephrectomies with an immediate mortality of 2 (*i. e.* 3·5 per cent.), (1 of sepsis, 1 of general tuberculosis), and remote mortality of 9 (*i. e.* 15 per cent.) The latter deaths resulted from tuberculosis of the lungs or other organs; two of these 9 probably had tuberculosis of the remaining kidney. Of the remainder, 41 (*i. e.* 71·9 per cent.) are "improved" or "cured." The former were those whose operation was most recent; pus was still present in the urine in 12 cases, and tubercle bacilli in 9 (guinea-pig test). Those who were not improved by operation numbered 4; 3 of these had developed tuberculosis of the remaining kidney.

Von Frisch's nephrectomies totalled 100. The deaths in the first 60 numbered 9, in the last 40 only 1.

Later deaths reached a total of 9; 6 of these occurred during the first three years after operation, the rest during the fourth and seventh years. The causes of death were pulmonary or visceral tubercle, general tuberculosis, and tuberculous meningitis. In addition one patient died (five years after operation) from tuberculosis of the remaining kidney. Details concerning the condition of the survivors are not given.

Mirabeau had performed nephrectomy 30 times—all on female patients. There were no deaths, immediate or remote, and in no case in which the second kidney was sound at the time of operation has it become tuberculous since. The exact condition of all these patients is not stated, but Mirabeau emphasises the importance of early operation before there is extensive bladder involvement. Of the 20 patients who had vesical tuberculosis at the time of nephrectomy there are only four in whom it can be said that complete restoration of the bladder has occurred. The rest have some degree of contraction and resulting irritability of that viscus; and in some cases also the urine remains cloudy from the presence of a secondary colon infection.

To sum up these results: When urinary tuberculosis is dependant upon a primary tuberculosis of one kidney, nephrectomy offers the patient a 60 to 75 per cent. chance of complete cure; as compared to certain death (probably within five years) if the kidney be not removed. The risk of operation is the immediate risk of any major operation, plus a small additional risk of general tuberculosis and tuberculous meningitis which obtains during the first year, and to a less extent during the second year, following operation. Good as these results are, they would be even better were the earlier reported cases excluded, for later years have given us improved methods of diagnosis, and experience has taught us which cases are unsuitable for operation.

In dealing with urinary tuberculosis the chief points to bear in mind are: (1) *That primary tuberculosis of the bladder is an infinitely rare disease.* Some cases of vesical tuberculosis are secondary to genital tuberculosis, but in the great majority of cases the primary seat of disease (so far as the urinary tract is concerned) is one kidney. This is an established clinical fact, so that we need not discuss theories or experimental possibilities in support of either a "descending" or "ascending" infection. Nor, from the practical point of view, is it a matter of vast importance whether the tubercle bacilli reach the kidney by way of the blood-stream, as is generally held, or through the lymphatics as suggested by Brongersma. In cases where vesical and genital tuberculosis are associated, we must remember that the seat of the primary disease may be the kidney also. Hanson (5) obtained genital tuberculosis after renal tuberculosis had been experimentally produced by inoculation of the renal pelvis. That the resulting genital tuberculosis occurred by way of the urinary stream, and not the blood-stream, was shown, first, by the fact that it resulted in males only, and secondly that it did not occur in these after ligation of the ureter.

(2) *Renal tuberculosis is at first a unilateral disease,* but post-mortem statistics show that the danger of the second kidney becoming tuberculous is very great if the tuberculous kindey be not removed. For instance, out of 191 post-mortem examinations done at the Allgemeine Krankenhaus, Vienna (6), on patients who died from renal tuberculosis, before the modern methods of diagnosis were in vogue, the disease was unilateral in 67 only. Possibly, as Israel suggests, the tubercle bacilli which have developed in one kidney have a special affinity for renal tissue, and are more likely to acquire a footing in the second kidney than in any other organ. This explanation scarcely seems necessary, however, when we consider that the bacilli circulating in the blood-stream are

excreted by the kidneys, and that the non-tuberculous kidney is in many cases already in a state of lowered resistance, from the presence of a toxic nephritis. *Nephrectomy, on the other hand, protects the second kidney from tuberculosis.* This is proved by operation statistics. In the above nephrectomy reports it will be noted how rarely tuberculosis of the second kidney was present at the time of death. It has been urged as an argument against nephrectomy that the post-mortem findings of unoperated cases so frequently show bilateral renal tuberculosis. It would be as logical to argue "this patient had syphilis, and died from tabes, therefore it is useless to treat syphilis." On the contrary it should be urged as one of the strongest arguments in favour of nephrectomy.

(3) *In renal tuberculosis the first subjective symptoms are usually vesical ones, and may occur while the bladder is still perfectly normal.* Symptoms pointing to the kidney are the exception rather than the rule. They occur in cases associated with some degree of obstruction (in the pelvis or ureter) and back-pressure on the kidney. It is by no means uncommon to find, at operation, a completely destroyed kidney, although the patient had experienced no renal pain whatever. Further, the tuberculous kidney is not necessarily an enlarged kidney; many kidneys which have been completely destroyed by tuberculosis are even smaller than normal. The infection of the bladder from the kidney may be a urine-borne infection; more commonly, however, it results from direct extension of disease; first, the kidney becomes tuberculous, then the ureter, then the bladder, so that by the time the patient comes under observation the cystoscope already shows, in that ureteric orifice, changes characteristic of inflammation of the ureter.

(4) *Latent intervals in the symptoms and apparent progress of the disease are common accompaniments of renal tuberculosis*, especially in earlier stages. The duration of such latent periods may be months or years. They may result from blockage of the kidney—cases are seen where complete obliteration of the lumen of the ureter has occurred—but more commonly the cycle of events is as follows: A tuberculous focus in the kidney softens, breaks down, and discharges itself into the pelvis; the orifice of communication becomes closed, so that the urine becomes clear again, the symptoms better—if tuberculous cystitis was present it improves or heals—and the patient may appear perfectly well, until the process, sooner or later, is repeated. This substitution of "an open" for a "closed" renal tuberculosis is doubtless the explanation of so-called cures by tuberculin.

(5) *The treatment of unilateral renal tuberculosis by tuberculin is not only useless, but dangerous* (though *after* nephrectomy it is invaluable in hastening the cure of the remaining cystitis). It is dangerous because the substitution of an "open" for a "closed" renal tuberculosis may lead to a false conclusion that the patient is cured. It is dangerous because it wastes valuable time during which the vesical disease is extending, and during which the genital organs, or the second kidney, may become tuberculous, and the time for operation slip past.

(6) When a diagnosis of urinary tuberculosis has been made, *the next step is to ascertain, firstly, which kidney is the source of the trouble; and secondly, whether the second kidney is an efficient kidney, and free from tuberculosis.* This information is obtained by means of the cystoscope. We need not discuss here the functional tests to be applied to the second kidney; by the time the patient has come under observation, this kidney has already taken on, at least in part, the function of both. Let us consider, on the other hand, what means we have at our disposal of ascertaining that this kidney is free from tuberculosis.

(*a*) The cystoscopic appearance of the ureteric orifice may enable us to say that the kidney is definitely tuberculous, but a healthy ureteric orifice does not necessarily mean a healthy kidney.

Cases are seen, for example, which at first sight appear to be instances of primary vesical tuberculosis. The cystoscope shows isolated or scattered foci of disease, but the ureteric orifices themselves, and the areas of bladder in their immediate neighbourhood, are perfectly healthy. When the ureters are catheterised, however, it is found that one or other kidney is tuberculous.

(*b*) Catheterisation of the ureter and examination of the separated urine for Koch's bacillus is the routine method to be adopted. It may be mentioned that a toxic nephritis of the non-tuberculous kidney, as evidenced by slight albuminuria, without the presence of casts or renal epithelium, is a not uncommon accompaniment of

tuberculosis of the opposite kidney. This may be disregarded, as it clears up after the tuberculous kidney has been removed.

(*c*) Guinea-pig inoculation is an additional safeguard when any doubt exists. Doubtless, cases occur in which microscopic examination for the tubercle bacillus is negative, and yet the inoculation test gives a positive result. On these grounds it might be argued that the test should be employed as a routine measure. In actual practice the margin of error is a very small one, and the six weeks' delay, which is necessary before the result of the test is known is often a serious disadvantage.

(*d*) The question naturally presents itself, *Is it possible to detect a "closed" focus of tuberculosis in a kidney which is apparently sound?* In such a case one might expect microscopic and inoculation tests to fail, but a new test has recently been introduced by Debré and Paraf (7) which renders the detection of such a "closed focus" possible. By this test the presence of the tuberculous "antigen" is demonstrated in the urine—the principles of the reaction being based on "complement-deviation."* Heitz-Boyer (8) reports 43 cases in which the test was tried; in all of these, save two, the result was confirmed by inoculation or subsequent examination of the specimen.

The value of the test (which failed in one case only) is shown by, *e.g.*, Case 36, in which a patient who suffered from albuminuria was found to have mononuclear cells present in the urine coming from the left kidney. Guinea-pig inoculation proved negative, but Debré and Paraf's test was positive. Subsequent nephrectomy proved the presence of tuberculosis of this kidney.

(7) *When the integrity of the second kidney has been proved, the earlier nephrectomy is performed the better will be the result.* Thus Wildbolz obtained cure in 76 per cent. of cases after early operation, whereas in late operations the number of cures dropped to 52 per cent. Delay involves the risk of secondary infection, but quite apart from this, it means more extensive vesical disease, and consequently a longer convalescent period. It means also a more difficult operation from perinephritis, and greater risk of infecting the peritoneum or operation wound with tubercle bacilli, or of producing generalised tuberculosis. But extensive vesical tuberculosis brings with it another danger, that is, the danger to the second kidney. So many cases are seen in which no tuberculous disease can be detected clinically apart from the urinary organs; in these cases, after nephrectomy has been performed, the bladder remains as the one source from which the second kidney may be infected. Delay also involves the risk of secondary genital tuberculosis, a complication which adds greatly to the gravity of the prognosis. Thus Wildbolz's statistics show that of the cases in which genital tuberculosis was already present, only 43 per cent. can be considered cured, and 27 per cent. have already died; whereas, of the cases in which the genital organs were not affected, 86 per cent. are cured and 6 per cent. only have died.

(8) *The contra-indications to operation* are few, but patients with visceral or pulmonary tuberculosis cannot be considered suitable subjects. The latter disease accounts for a large proportion of the deaths occurring in the first two years after operation. Genital tuberculosis, though adding to the gravity of the prognosis, cannot be considered a contra-indication. It must be remembered that this is a much more chronic form of tuberculosis and can be successfully treated with tuberculin, after nephrectomy. Extensive bladder tuberculosis likewise cannot be considered a contra-indication to operation; although in these cases the end-results are much less satisfactory, yet nephrectomy offers the patient his one chance of cure, and even at the worst, some amelioration of his symptoms.

In cases of bilateral renal tuberculosis the question of operation does not often arise, but even here, when early disease is present on one side, nephrectomy may be indicated on the opposite side. Such indications would be a functionless kidney, or one which was the seat of severe pain or hæmorrhage.

* The urine of tuberculous patients who have no urinary tuberculosis may contain antibodies capable of producing this complement-deviation. These are destroyed by heating the urine to 60°.

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(1) "3e Kongress der Deutsch. Gesellschaft für Urologie," 'Zeitschrift für Urologie,' January, 1912.

(2) Phillipowicz.—'Wien. Med. Blätter,' 1883.

(3) Pousson.—"Diagnose de la Tuberculose Rénale," 'VIIIe Sess. de l'ass. française d'Urologie,' 1904.

(4) Thévenot et Batier.—"De la bacillurie tuberculeuse," 'Province Med.,' 1907.

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(6) Quoted by Alexander Homburg, "Zur Diagnose und Therapie der Nieren- und Blasentuberculose" 'Dissertation, Freiburg i Br.,' 1911, S. 73.

(7) Debré and Paraf.—'Soc. de Biologie,' July and October, 1911.

(8) Heitz-Boyer.—'Journal de Urologie,' January, 1912.

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A MEETING OF THE HUNTERIAN SOCIETY.

PRESIDENTIAL ADDRESS

ON SHOCK,

By A. H. TUBBY, F.R.C.S.,

FOLLOWED BY DISCUSSION ON

INTRA-VENOUS ANÆSTHESIA.

THE President thanked the Fellows for the honour conferred upon him, which allowed him to follow in the footsteps of so many distinguished men, all of whom testified to the genius of the great John Hunter, and of his scarcely less distinguished brother, William Hunter.

Continuing, Mr. TUBBY said: During the summer another great man passed away—Lord Lister—and those who were present in the Abbey will never forget the solemn majesty of the funeral service and the great gathering of representatives of science from all parts of the world. Lister was primarily a physiologist, then a pathologist, and finally a surgeon, who sought the opportunity to put his earlier researches to the test of experience. It is to his great work and untiring perseverance that we owe the greatest advances in surgery since Hunter's time. Following in Lister's footsteps there are numerous workers in the physiological field, who have taught us how to avoid dangers other than septic; and it is upon some of the recent work and the application of some physiological investigations to surgery that I propose to speak very briefly to-night.

Surgical shock.—Whilst the surgeon has been enabled to abolish sepsis and the anæsthetist to subdue pain, shock still remains one of the great barriers to perfect success. According to Crile and Mummery shock is due to exhaustion of the vaso-motor centre in the medulla; it appears that some

of its chief causes are interference with, bruising, cutting and crushing of, the nerves, exposure of, handling and dragging upon the abdominal viscera, and the loss of carbonic acid gas. The effects of these stimuli are to exhaust the activity of the vaso-motor centre and cause dilatation of the small arteries, especially in the abdominal area. The result is that not enough blood is sent to the vaso-motor centre.

The *avoidance* of shock is all-important, and Rendle Short has pointed out that in the case of operations involving big nerves, such as the sciatic and anterior crural, a preliminary injection of five minims of novocaine solution prevents a great deal of shock. It may also be avoided by intra-venous injections of saline, by bandaging the extremities so as to prevent dilatation of the peripheral arterioles, and Crile accomplishes the same end by enveloping the patient in a pneumatic suit under pressure. Rapidity of operation and an equable warm temperature of the room are also important. Recent research has shown that excessive excretion of CO_2 is an important factor in the production of shock.

To *relieve* shock we may use a solution of adrenalin of a strength of 1 in 50,000, and a very valuable remedy is pituitrin, or extract of the pituitary gland. It is necessary to repeat the injection of the latter in about two hours, as the effects pass off rapidly. The head should also be placed low and the extremities raised. Strychnine is of doubtful value, and alcohol is no good whatever.

Acapnia (carbon-dioxide hunger) and shock.—According to Haldane and Priestly, excess of CO_2 in the blood is the great stimulant of the respiratory centre. When the amount of CO_2 falls below the normal, shallow breathing and a quick heart beat occur and blood-pressure falls. Further diminution of CO_2 produces another striking effect. Exudation of plasma takes place from the vessels, and this may explain some cases of œdema of the lung after anæsthetics. Therefore it is essential to maintain, if possible, a proper proportion of CO_2 in the body. Excessive struggling during the early stages of anæsthesia is to be avoided, as much CO_2 is exhaled, and the respiratory and vaso-motor centres lack their natural stimulus.

The effect of loss of CO_2 on the intestines is striking: they rapidly lose their peristaltic action. Further, when the peritoneum is opened a great deal of CO_2 escapes from the intestines and the small vessels become injected and dilated, and this causes shock.

The loss of CO_2 may be *avoided* by administering the anæsthetic through a long tube so as to increase the "dead breathing space," and by avoiding spasm during inhalation of the anæsthetic. The loss of CO_2 may be counteracted by injections of saline solution, saturated with carbonic acid, into the veins, or by pouring this fluid into the abdominal cavity and closing it quickly. In abdominal operations it is important that the intestines are exposed as little as possible; and if they be, they should be covered with large sponges wrung out of hot saline solution into which CO_2 has been passed.

Another most important application of physiology to abdominal surgery has resulted in an extraordinary diminution in the mortality from peritonitis, after operations, in recent years. Whereas, ten years ago, in gangrenous appendicitis with general peritonitis the mortality was 90 per cent., it has now been reduced to under 10 per cent., and this is due to the laying down of certain postulates of treatment by Murphy, of Chicago. These are—(1) The semi-upright position after operation. It has been shown that the absorptive powers of the upper part of the peritoneal cavity are many times greater than that of the lower part, the pelvis; and therefore fluid should be allowed to drain downwards; this is effected by the semi-upright position. (2) Rectal infusion: the object of this is to get rid of the fluid accumulated in the pelvis. This is accomplished by passing into the rectum. by means of a suitable apparatus, ten to twenty pints of saline solution at a temperature of 108° F. The fluid is absorbed into the pelvic peritoneum and escapes through the open abdominal wound. This is the "sheet anchor" of abdominal after-treatment. And the speaker added that he had seldom known a patient to die if after an abdominal operation twenty pints of saline fluid had been absorbed. (3) One great danger incidental to abdominal surgery is intestinal paralysis; opium, therefore, should be strictly avoided, and salicylate of eserine should be injected to increase peristalsis. Pituitrin also has the same effect, and further, it diminishes shock by contraction of the intestinal vessels. (4) Lastly, in order to get rid of poisonous intestinal contents small doses of calomel should be given frequently.

Mr. E. G. SCHLESINGER said the intra-venous method of giving anæsthetic drugs has a very definite, but limited application; indiscriminate use of the method is to be deprecated. The indications for giving intra-venous ether are, briefly, cases of severe shock (*e. g.* peritonitis), cases of hæmorrhage (*e. g.* ruptured ectopic), cases with lung complications, and cases where the patient is very nervous (*e. g.* exophthalmic goitre). The most perfect aseptic technique is essential, and too great attention cannot be paid to this, since a single case of sepsis would be an unjustifiable calamity.

In order to secure rapid induction a large-bore cannula is needed, which necessitates cutting down on the vein. The apparatus used is simple, consisting of a reservoir, a combined dropper, bubble-trap and warming chamber, and a cannula. Too great attention need not be given to the temperature at which the solution is used, providing only that it never exceeds 99° F., at which temperature the ether evaporates. A 5 per cent. solution of ether in normal saline is used, and this is mixed cold. It is practically impossible to give an overdose if the patient is breathing, and the method can readily be employed single-handed. The advantages of ether over hedonal are that the patient comes round very rapidly, that ether is excreted by the lungs, and that ether causes much

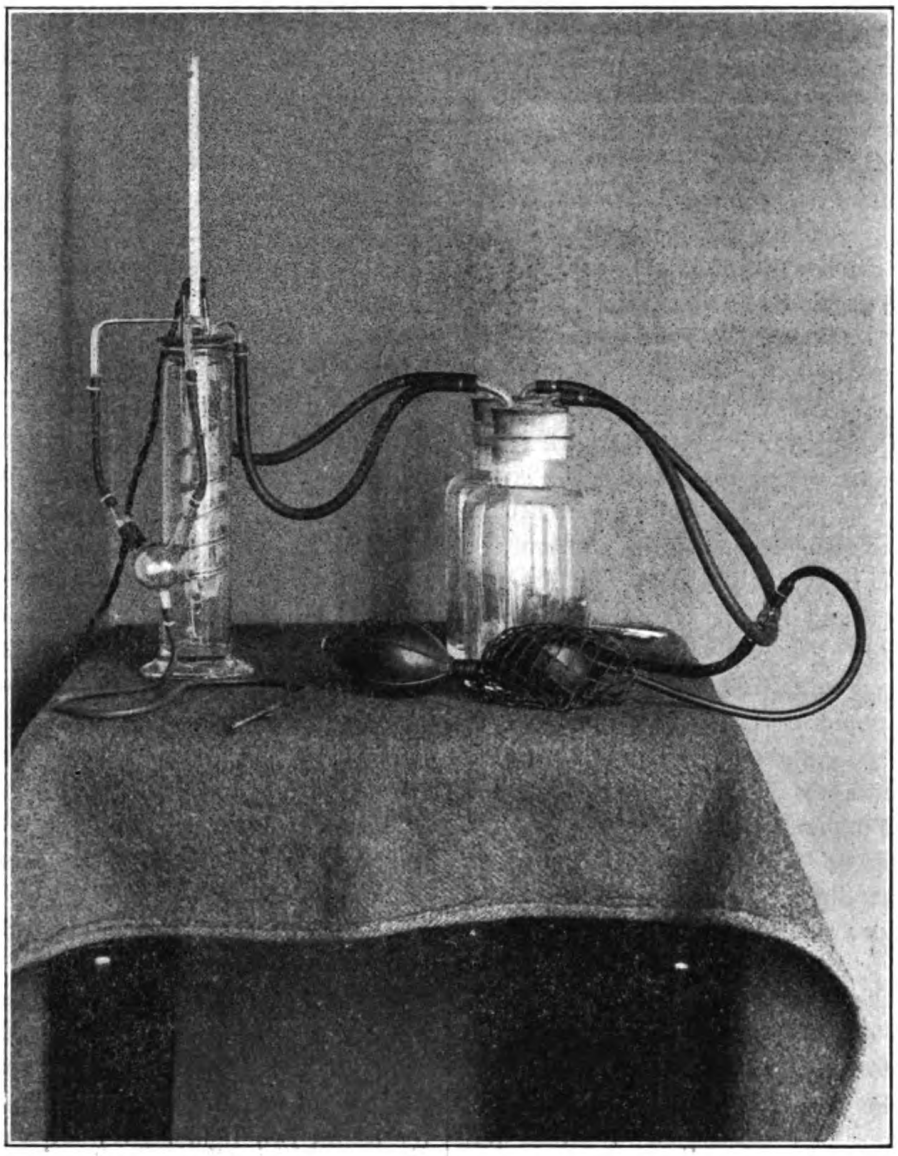

FIG. 1.

less bleeding than hedonal. No vomiting occurs with this method, and thrombosis should never take place, it being invariably due to faulty technique. The apparatus used and the technique of the administration have been fully described elsewhere.*

Mr. H. S. SOUTTAR (London Hospital) demonstrated an apparatus (Fig. 1) for the intra-venous administration of anæsthetics, which consists of a reservoir, a pressure apparatus and a heating chamber. The reservoir is formed by two glass bottles, each holding 750 c.c., with rubber stoppers through each of which two tubes pass. Two of these merely pass through the stoppers and are together connected with the rubber bulb which forms the pressure apparatus. By this means the liquid in the reservoir may be placed under any desired pressure. The other two tubes reach to the bottom of the bottles, and serve to carry off the liquid to the heating chamber. This is a glass cylinder filled with water at a temperature of 100° F. In it are two coils of pure tin tube through which the liquid flows, and a coil of nickel-iron wire enclosed in a glass tube and carrying a current from a cautery circuit. The current can be controlled by the cautery resistance and maintains the temperature constant at 100° F.

The two delivery tubes from the heating chamber discharge their contents through separate nozzles into a common bulb of glass. From this a small rubber tube passes to a hollow needle which is inserted into the patient's vein. The bulb acts as a mixing chamber and an air-trap. In the event of much air or vapour collecting it can be allowed to escape through a small aperture in the top of the bulb.

The needle used is a large hypodermic needle, and is inserted directly through the skin into the vein. In rare cases a small incision may be necessary to expose the vein. We consider this method far superior to that of dissecting out the vein and tying in a cannula. The necessary dexterity is soon acquired.

The apparatus can be used for the injection of any intravenous anæsthetic, but is specially intended for ether. The addition of a little alcohol (2 per cent.) allows the use of a 10 per cent. solution of ether in one of the bottles, the other being filled with pure saline. An equal rate of flow from the two bottles gives a 5 per cent. solution, and as it is under constant pressure no bubbles are formed, though it is delivered at 100° F. The double reservoir is a great convenience, as one bottle may be disconnected and refilled without stopping the flow, whilst the anæsthetic can be diluted to suit the circumstances of the case. The double reservoir has the further advantage that it enables one to start with one anæsthetic and to pass continuously to another. It is therefore particularly suited for the induction of anæsthesia by paraldehyde and its continuance by ether or hedonal, a great saving of time being thereby effected. Regurgitation of liquid from one bottle to the other is absolutely prevented by the double dropping bulb.

As a variation we have heated the reservoir bottles by surrounding them with hot water, and suspended the tin coils in a Thermos flask containing water at 100° F. The flask acts as a thermostat, and adjusts any variations in the temperature of the liquid delivered to the tubes.

Mr. H. L. C. NOEL (London Hospital) spoke of the use of paraldehyde as an intra-venous anæsthetic. He said that in the administration of specific drugs whose action is general and not local, the modern tendency has been to employ intravenous injection in preference to other methods. Immediate and accurate results are thus assured. Paraldehyde is a specific hypnotic, and the results of many years of use tell us that it is safe, and can be used in extremely grave cases, but its taste is so unpleasant as to constitute a serious drawback. Under these circumstances we decided to give it by intra-venous method.

Starting at first with extremely small doses we injected 4 c.c. of the pure drug into the median basilic vein. Almost before the syringe was empty the patient was unconscious. He slept naturally for about two hours. Subsequently 8 c.c. were injected with satisfactory results. We found, however, that the pure drug caused a certain amount of local discomfort and coughing. We therefore diluted it with saline to form a 10 per cent. solution. We find that 100 c.c. of this solution produces in about forty seconds an anæsthesia lasting about twenty minutes, and suitable for minor surgical procedures. Many attempts have been made by Burkhardt and others to find a drug capable of rapidly inducing anæsthesia as a pre-

* 'Guy's Hospital Gazette,' May 25th, 1912.

liminary to intra-venous ether. Of these isopral was the most satisfactory, but was not very fast. We therefore decided to use paraldehyde for the same purpose, and have found it most valuable, combining as it does rapidity with safety. Paraldehyde, whilst acting temporarily as an anæsthetic, has a prolonged hypnotic effect, considerably reducing the amount of ether subsequently required. Being volatile it is rapidly excreted by the lungs —an important element in the safety of the drug and an advantage which cannot be claimed for hedonal.

Mr. C. M. PAGE said: The induction of general anæsthesia by means of the intra-venous infusion of hedonal has now been practised for some eighteen months. I propose to outline the conclusions at which I have arrived during the last year.

First, in regard to the technique, at St. Thomas's Hospital the method I described in my original paper is still employed. Infusion is usually made into the internal saphenous vein, where it lies on the internal malleolus; no particular fixation of the leg is necessary. I prefer to expose the vein by incision rather than to puncture the skin and vein with the cannula.

As to the dangers of the method, several fatalities which have been recorded have been, in my opinion, due to an overdose of the drug; I think that there should usually be some reaction on the part of the patient to the skin incision; if this is expected and allowed for there is usually no further trouble, as such a degree of anæsthesia is not associated with rigidity of the muscles or any deep reflex.

Local thrombosis occurred in rather a large number of cases I had under observation in the summer; this I think was due to the practice of putting the solution into the tank at a temperature of 120° F.; since a solution at 105° or 110° F. has been used I have not noticed the complication. Cases which in general seem unsuited to the method are those of acute lung disease, general arterio-sclerosis, or cases where the heart is not sound. I do not think old people are suitable subjects, and in operations in which there is a liability to post-operative bleeding into the mouth and air-passages I do not advise this method of promoting anæsthesia.

Cases in which I think the method has proved strikingly useful are cerebral and spinal cases (including the exophthalmic type) and cases of intra-abdominal hæmorrhage.

In cases of extensive cellulitis of the neck a light anæsthesia in which the deep reflexes are not abolished can very readily be obtained; in fact this is the only way I know of in which operative measures can be safely carried out for such conditions without hurting the patient.

Dr. SILK was of opinion that the demonstration of the possibility of producing narcosis by the intra-venous method was one of the most notable advances that had been made in the department of anæsthetics for many years. But what was possible was not always expedient, and he was glad to find that the previous speakers had recognised that this method had its limitations. He thought that Mr. Page's summary of the cases suitable or unsuitable for hedonal was a good one, and applied equally to ether. The method, too, was complicated, and involved the presence of an assistant to the anæsthetist, which militated against its routine adoption.

The novelty of the apparatus shown that evening seemed to consist mainly in the means adopted to warm the inflowing stream of fluid. He thought that this point had been rather over-elaborated. In actual practice he had not noticed any evil effects following the injection of a fluid at a temperature of between 75 and 85, *i.e.* but little over that of the operating room, and this could be done by simply coiling the delivery-tube in a basin of warm water. He thought that a glass cannula was preferable.

He was glad to agree with Mr. Page that many of the fatalities recorded against hedonal were the result of over-dosage. The strength of the solution originally recommended was ·75 per cent, which worked out at something over 65 grains to the pint, which he considered too strong a dose.

Mr. H. E. G. BOYLE said: Of anæsthesia produced by the intra-venous injection of hedonal I have no personal experience, and so I can only speak of it from what I have heard and read. There is one point, however, which has seemed to me to be not altogether in favour of the method. I mean the long period of unconsciousness that seems to follow the anæsthesia. This cannot, I think, be altogether a desirable effect. I know quite well that we shall be told that it is a good thing for the patient to be asleep or unconscious,

as the case may be, during the time that he is likely to feel pain from his operation, but is this always a good thing? I think not.

There are certain cases in which the pain after the operation is considerable, but on the other hand there are many operations in which the after-pain is but slight, and I cannot see that it is advantageous to have these patients unconscious for several hours after the operation. I imagine that these unconscious patients require very careful watching until they regain their conscious state—this alone entails added responsibility, and possibly danger.

But there is one point that I particularly wish to emphasise to-night, and it is with regard to the so-called advantages of hedonal and ether infusion anæsthesia over inhalation anæsthesia for large operations about and in the mouth.

Mr. Rood, quite recently, in his paper at Liverpool, speaking of ether infusion for this type of case, said, "In operations upon the mouth, jaws, or pharynx, the difficulties of the surgeon, in the nature of the case quite great enough, are much increased by his having to share the field of operation with the anæsthetist; and it not infrequently happens that the surgeon or the anæsthetist has to be content with a less degree of technical perfection than he would have obtained if he had not been embarrassed by the necessities of the other."

Now, sir, although this description is doubtless true, I do not for one moment agree that it is at all necessary. I think that in a mouth case, when sponging the throat is necessary to preserve a patent air-way, that the anæsthetist is the man to do it. He knows when there is danger to his patient from blood or other matter getting into the larynx, and sponges it away. The surgeon or his assistant does not usually do this; they are frequently too engrossed in their difficult task to bother about the anæsthetist and his troubles.

In ether infusion, if the anæsthetist is to be attending to his infusion, who, then, is to look after the patient's larynx to see that it does not get filled with blood? I claim that there is a better and safer way out of the difficulty. Some years ago Sir Henry Butlin taught us the value of performing a preliminary laryngotomy and packing the pharynx when doing these major operations in the mouth. To Butlin's laryngotomy tube I have added an extension tube that fits accurately into the laryngotomy tube, and is connected by a rubber tube to the cone of a Hahn's inhaler; with this extension tube in position the anæsthetist is removed from the field of operation, and can devote his attention entirely to the anæsthetic—there is no fear of blood getting into the larynx, since this is securely packed off. I feel confident that anyone who has performed these large throat operations with the anæsthetic given as I have described will infinitely prefer the method to any other on account of its perfect safety, and on account of the quiet anæsthesia that is obtained.

I have been giving anæsthetics long enough now to have seen various anæsthetic agents and methods brought forward and hailed as *the perfect anæsthetic* that we had all been looking for, and yet some have dropped out of use, and the others have been relegated to their proper place amongst our list as suitable for certain cases.

There is a danger, whenever a new method is advanced, of enthusiasts being found who will advise it for everything, and I should like to-night to sound a note of warning with regard to intra-venous anæsthesia. I do not believe that in either of the substances in use at present we have found the perfect agent; that each of them is useful in certain cases I freely admit, but we should be careful lest in our zeal for new and advanced things we do not rashly advocate these methods when the older ones might be more suitable. Let us rather welcome them as additions to our armamentarium, and reserve their aid for those cases to which they are particularly suited.

Mr. Ashley Daly said: I have not much to add to what has already been said, as my experience of intra-venous anæsthesia is small and is limited to a few cases of hedonal; but I have had the privilege of helping Mr. Souttar and Mr. Noel with some of their paraldehyde and ether cases, and have seen the hedonal method carried out a good many times by others.

One of my hedonal cases was a very feeble woman of seventy, with chronic bronchitis, who underwent an extensive operation for the removal of carcinoma of the breast, and this, to my mind, is the class of case where infusion is useful, hedonal rather than ether; and I think infusion should be confined to those cases with lung disease and to those who require saline solution, such as a ruptured tubal gestation.

I quite agree with Dr. Silk and Mr. Boyle that it is inadvisable to use this method in jaw, mouth and throat cases, as the bleeding will be more severe and the anæsthetist will be unable to give his whole attention to keeping the throat and larynx free from blood, owing to his having to regulate an apparatus at the patient's foot or arm; and I think that in a formidable mouth operation a much more satisfactory result will be obtained if the operator first does a laryngotomy, and packs off the pharynx so that the anæsthetist may administer chloroform through the laryngotomy tube with a Junker's inhaler.

I cannot see that, except the two classes mentioned above, the infusion method offers any great advantage over the inhalation method, especially over open ether preceded by morphine and atropine, which is the safest and most satisfactory anæsthetic and is applicable to almost any case, and which gives rise to few unpleasant or dangerous after-effects. Mr. Page recommends hedonal in head operations, but I have lately taken to using open ether for these with excellent results, and for my last three anæsthetics for excision of the Gasserian ganglion I have used open ether throughout, and in each case the operator has remarked on the almost entire absence of bleeding, which is generally such a troublesome feature is these cases.

December 30th, 1912.

Reduction of Fragments in Fractures.—Two methods for reducing fractures are described by Gerster. One is a traction method in which he separates the fragments forcibly by means of a special apparatus (hook and chain) which overcomes overriding and makes accurate approximation possible. The other is a reduction method. After proper exposure and freeing, a Lowman clamp is applied to each fragment. A turnbuckle is so inserted that the jaws at its ends engage the shafts of the clamp on either fragment. As the barrel of the turnbuckle is rotated, the clamps (fragments) are forced apart. When the turnbuckle has reached its maximum extension, a second longer turnbuckle is applied, and continues the extension as far as necessary. The turnbuckle's jaws are made loosely to fit the shafts of the Lowman clamps in order to permit the fragments (which are out of line) being gradually brought into their proper relation.—*Journ. A.M.A.*, vol. lix, No. 22.

A POST-GRADUATE DEMONSTRATION

ON THE

DISEASES OF CHILDREN.

At the Salford Royal Hospital.

By HUGH T. ASHBY, B.A., M.B.Cantab., M.R.C.P.Lond.,

Assistant Physician to the Hospital; Physician to the Manchester Children's Hospital, Pendlebury.

GENTLEMEN,—I have collected to-day a number of children who show a variety of diseases all fairly commonly met with in practice, but at the same time interesting in themselves. The first two cases, æt. respectively 8 and 9 years, are suffering from congenital heart disease. In neither case is the diagnosis obvious at sight as it often is in these cases, and it is only after we have made a careful clinical examination of the children that we can be sure they both have a congenitally malformed heart. There is no cyanosis and also no clubbing of the fingers—two symptoms which go hand in hand, as those cases which have marked cyanosis generally have clubbing of the fingers and toes due to a back-pressure on the venous system. Now it is well to remember that rather less than half of these cases ever have cyanosis at any time, so that the absence of this sign does not in the least negative the diagnosis. Both the children are well built, and neither has at present much bronchitis—two satisfactory symptoms, as these children very easily catch cold, and at the same time very quickly begin to lose weight if anything goes wrong with them. Taking the girl first, you will find that there is a very marked thrill over the base of the heart, a sign I think which almost makes the diagnosis certain, as the thrill felt in ordinary acquired heart disease in a child is limited to the apex of the heart. She also has a very marked murmur best heard over the tricuspid area and widely conducted, especially upwards, and it is very rare for the tricuspid valve to be affected alone in a child with acquired heart disease. Still another fact in favour of this being congenital in origin is that the mother will tell you that the child often has attacks of pain over the heart lasting a few minutes; this also is a rare symptom in acquired heart disease in children, except when the heart is working very badly and the child almost *in extremis*.

The other child also has a thrill over the base of the heart, and a very rough systolic murmur best heard over the pulmonary area and best conducted upwards. As regards the treatment of these cases, we cannot, of course, do very much to the heart itself, but we can do a very great deal by very carefully looking after these children, and the better this is done the longer will they live. They very easily catch cold, and they feel the cold very quickly, and on this account they must be kept warmly clothed and not allowed to go out in any cold or foggy weather as we so often experience. It is as well to weigh them regularly, so as at once to detect any loss in weight, which will show that the heart is not quite keeping up to the demands made upon it. Children with acquired heart disease very quickly lose weight if they are not cared for, but these cases lose weight even more quickly. If they are liable to attacks of cyanosis it is as well to always have at hand some amyl nitrite capsules ℿij, which can be used directly an attack comes on, and in this way shorten it and relieve the child. Both these children are being very carefully cared for, with so far a very satisfactory result. We can, of course, only guess at the lesion, but whatever it is, it is not of the most serious type.

The next case is a girl, æt. 13 years, whose neck has begun to grow larger for the last six months. The enlargement is due to a diffuse bilateral hypertrophy of the thyroid gland. It has caused the child no inconvenience, and the mother would not have brought the child except that the neck is gradually and steadily getting larger. If you will examine the child you will find none of the signs of Graves's disease; there is no exophthalmos, no tremor, no tachycardia, etc. The swelling is a simple physiological enlargement of the thyroid gland, which is often seen about puberty, and it is probable that the body calls for more thyroid secretion at this age for some reason, and that the gland enlarges to supply this want. In support of this view is the fact that if we give this girl thyroid extract by mouth the enlargement of the gland will go down, on account of the artificial thyroid taking the place of the hypertrophied gland. It is as well to continue for some time with small doses of the thyroid extract after the swelling has gone down, and I do not think these cases ever go on to Graves's disease.

This is a girl, æt. 5 years, who last July (five months ago) had a convulsion, and what the mother calls a bilious attack, lasting for three days, at the end of which time the mother noticed that the child was paralysed all down the right side. The convulsion came on suddenly and without any exciting cause as far as can be made out. When brought to the hospital there was a complete hemiplegia, with increased reflexes on the affected side, showing that the lesion is in the brain and not in the spinal cord—in fact, the child has had an acute encephalitis in the cortex of the left side of the brain. The ætiology is most likely the same as that in acute anterior poliomyelitis, except that the damage has been done in the motor area of the brain instead of in the spinal cord. The child has gone on quite well, and, as you can see, the leg has completely recovered, but the arm is held flexed at the elbow, and very little power has come back as yet. This is the usual course in these cases; the leg completely recovers, or nearly so, but the arm is left weak, and but of little use. She is having regular massage to the arm to prevent contractures, and she wears a splint on the forearm and hand at night. She is encouraged to use her arm, and to help her to do this the mother ties the sound arm to the child's side for a short time each day.

The next case is one of spastic diplegia. Unlike the former case, the lesion here is ante-natal, and most likely due to a hæmorrhage into the cortex of the brain at birth, or else to a degeneration of the cortex taking place in intra-uterine life. In these cases the mental condition is generally bad, but in this case it is quite good, and for this reason more amenable to treatment. To overcome the spasticity of her legs she has had an operation. About two months ago Mr. C. Roberts did a laminectomy, and divided the posterior lumbar nerve-roots, and by this means the reflex arc was divided and the spasticity overcome, as you can see by the way the legs are easily flexed and extended. Before this operation her legs were extremely spastic, the knees were held flexed, and the thighs rotated inward and adducted; in this condition it would be of no good trying to teach her to walk, or even to stand properly. By dividing the posterior nerve-roots the child has, in fact, had locomotor ataxia given her, and she is being re-educated to use her legs on the same lines as a

case of locomotor ataxia, *i. e.* re-educated. She can now stand by herself, and she is just learning to go about by holding on with her hands, and in time I hope she may learn to walk without help. The teaching will need a good deal of patience and perseverance, but it will be well worth while doing, and, indeed, as she is now, she is in a better condition than she was before the operation. It is of no use doing this operation on a child who is mentally defective, as the necessary teaching afterwards cannot be carried out effectually.

This is a baby, æt. 1½ years, and what is called a Mongol. The facies is characteristic, and, as the mother will tell you, the neighbours call it the Japanese baby, which very thoroughly describes it. The history of these cases is often the same, and they are generally the last of a large family, or else there has been a long interval between the Mongol and the child born before it. In this instance the child born before is now twenty years of age, so that there was an interval of over eighteen years before this child. You will notice the small round head, the eyes sloping downwards towards the nose, and the very marked epicanthic folds of the inner sides of the eyes. The hands are small and podgy, and are what are called the trident hands; the little finger is short and curved and does not reach to the distal interphalangeal joint of the ring finger. These children are always backward and they seldom reach adult age. As babies they are easily looked after, but they do not come on as they should, and they always remain as children and rarely learn to read or write. Some of them improve a good deal with thyroid extract given for a long time, but to not nearly the same extent as a cretin. They all seem to have a very small naso-pharynx, and on this account any small amount of adenoids causes them a good deal of trouble, and they all seem to suffer in this way at some time of their lives.

This is a girl, æt. 10 years, who was brought by her mother on account of appearing pale, although there was nothing else noticed wrong. Now in judging anæmia the personal element comes into play a good deal, and errors of judgment are easily made. A child may appear pale, but in reality is perfectly normal, and the only real way is to make a blood examination, which not only tells us if there is anæmia, but also the amount of anæmia there may be. The complexion of the child often makes it difficult to say if there is any anæmia; thus fair-haired children with a light complexion appear pale, like this child, without there being any anæmia, and any slight amount of anæmia there is becomes exaggerated to the eye. Anæmia is, however, commoner in blondes than in others. On inquiring into the history of this case, we find that the child has always been this colour, and the mother herself was exactly the same when younger, and this tendency very often runs in families without being of any significance. This case, then, just shows that the pallor is perfectly normal and needs no treatment, and we should only be wasting time in giving her iron, and we should not by any means improve her colour. The pallor may be due to some peculiarity of the skin, or perhaps to an alteration in the distribution of the blood, there being but little blood circulating in the skin. Europeans who have lived in the tropics become pale and remain pale, but they often have no true anæmia as in this case.

December 30th, 1912.

Sign of Pericardial Effusion.—Maragliano remarks that all writers agree that the effusion must be of considerable volume to be evident on Roentgenoscopy; the curve of the heart outline is changed from normal, the shadow assuming more a triangular shape and the pulsation of the heart becoming less evident. The pulsation of the lower margin of the heart is transmitted to the air-bubble in the stomach in normal conditions, but when there is an intervening accumulation of fluid in the pericardium only a minimal pulsation is transmitted from the right ventricle. The findings are more pronounced if the air-bubble is rendered conspicuous by taking an effervescent powder. Five cases are described in which the lack of a transmitted pulsation in the air-bubble in the stomach confirmed the assumption of an effusion in the pericardium, and puncture or necropsy revealed the fluid. The sign disappeared as the patients recovered. In three other cases in which a pericardial effusion was suspected, the marked pulsation in the stomach-bubble testified to the absence of an effusion in the pericardium, and this was confirmed by the course of the cases. In dilatation of the heart without effusion there was always more or less pulsation.—*Journ. A.M.A.*, vol. lix, No. 22.

A CLINICAL LECTURE

ON

THE RATIONAL TREATMENT OF VENEREAL DISEASES.

Delivered at the Polyclinic.

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GENTLEMEN,—I want to-day to give you some outlines of the treatment of venereal diseases in general, and I propose to begin by saying a few words about gonorrhœa. Let us take first an ordinary case of acute gonorrhœal urethritis. The patient, we will suppose, comes to you with an attack of gonorrhœal urethritis of only two or three days' duration, then it is well to try the abortive treatment upon him. But you will only try that supposing there are no subjective symptoms: in other words you will not attempt the abortive treatment if the patient has œdema of the penis, or is passing blood, or has very great pain on passing water, and it is no use attempting it if the urethritis is of more than two or three days' standing. The abortive treatment consists in flushing out the anterior part of the urethra two or three times a day with about a pint of a warm 1 per cent. solution of hegonon, or a 5 per cent. solution of protargol, or a 25 per cent. solution of argyrol. Do not carry on this procedure for more than three days, and if the discharge has not stopped by the third day the ordinary treatment for acute anterior urethritis should be substituted. If the abortive treatment has been of any use, by the end of the third day no gonococci should be found.

The best way to treat ordinary acute anterior urethritis is to give the patient a wash-out once or twice a day with a 1 in 4000 solution of potassium permanganate. As a rule a case of acute anterior urethritis is unaccompanied with complications, so there is only the local treatment to attend to.

Next with regard to the patient who has chronic urethritis; and I should like to remind you that over three-quarters of the cases of acute urethritis become chronic if treatment by injections is not started early, and as chronic urethritis in most cases is associated with prostatitis, and as complications are very common when that gland is involved, special treatment is required. For an ordinary case of subacute or chronic posterior urethritis, in which the prostate is more often than not associated, you should massage the prostate every other day and give an instillation by means of an Ultzmann's syringe of a 0·5 per cent. solution of silver nitrate, and in addition advise the patient to syringe himself night and morning with potassium permanganate 1 part in 4000, or with a 0·1 per cent. solution of albargin.

I should now like to call your attention to a very important point in connection with chronic urethritis. In a number of cases the condition, to all intents and purposes, disappears, and the patient, thinking he is cured, has connection, with the result that his discharge starts again. When this happens it is nearly always diagnosed by medical men as a second, a third, or a fourth attack of gonorrhœa, but wrongly so, since almost all these further attacks are cases of recurrent urethritis, the seat of the origin thereof being usually the prostate, the prostatitis not having been diagnosed and therefore not specially treated when the patient first came under observation. A recurrence, in contra-distinction to a fresh attack, is a very important point to remember, not only from a diagnostic point of view and the treatment suggested thereby, but also from a point of view of prognosis, especially when marriage is contemplated, because it is easier to cure a case of anterior urethritis than a chronic prostato-urethritis. If a patient has had chronic urethritis and has no discharge, a bacteriological examination of the prostatic secretion will seldom give a definite indication as to whether that patient is cured or not. The best way to ascertain whether the patient is cured is to give him a subcutaneous injection of a very large dose of vaccine, say 50 to 100 million of a potent vaccine, when, within forty-eight hours following the injection, if the patient has a hidden prostatitis or chronic posterior urethritis, he will get an increase in the morning drop or even a profuse discharge.

Now a word about complications. The complications of gonorrhœa are nearly always associated with either a chronic posterior urethritis, or more often with a chronic prostato-urethritis; therefore it is no good treating the complications alone unless you treat the local condition as well. That is to

say, if you employ vaccines for the treatment of the complications, the vaccines may completely cure the complications, but they will seldom cure the prostato-urethritis; and if you leave the prostato-urethritis, the seat of the whole mischief, untreated, it will almost certainly give rise to trouble later, and the complications will recur. First of all let us consider epididymitis. If the condition is unilateral the best method of treatment is to put the patient to bed and rely upon the frequent applications of evaporating lotions to cause resolution. The diet should be a light one, and sodium salicylate should always be used internally in preference to antimony. If the epididymitis is bilateral and there is therefore a risk that the patient may become sterile, it is necessary to adopt active measures. The best treatment is to take the testicle in your hand, get the epididymis uppermost, and plunge a scalpel or sharp bistoury into it in two or three places. The procedure is a painful one, but the pain is fully compensated for by the immediate relief which ensues. The reason why a patient becomes sterile from bilateral epididymitis is because the gonococcus has a greater tendency to give rise to the formation of fibrous tissue than pus, and as the fibrous tissue contracts it occludes all the seminal tubules.

Consider arthritis next. Arthritis may be acute, or it may be chronic; many of the acute cases get well of their own accord, but most of the chronic cases are not only troublesome to treat, but may be very difficult to diagnose. Because vaccine treatment is practically the only method we have of satisfactorily dealing with chronic arthritis, and because I have recently done some research work along these lines in conjunction with Dr. B. G. Klein, you will allow me to make more than a brief reference thereto. I divide vaccines into three categories: the ordinary vaccines with which you are familiar, and most of you have no doubt used; the intra-venous injection of the autolysed vaccine; and the sensitised vaccine.

I will first mention the ordinary vaccine, but before going any further must warn you to exercise great care in the selection of your vaccine, since the stock vaccines are usually of no use whatever—a fact you will appreciate when I inform you that a gonococcal vaccine begins to deteriorate after fourteen days, even if kept at a temperature of zero and away from the light.

If your vaccine is a potent one (the potency can be gauged by the antigen properties) it has no need to be autogenous; your initial dose should not be more than from three to five millions, and when giving this injection always use the muscles as the site for injection rather than the subcutaneous tissue, as many a severe and painful local reaction may be thereby avoided. After an injection the patient gets an aggravation of his symptoms, and either an increase of the morning drop or an acute discharge; that is to say he has a negative phase and is ill; and then in two or three days' time the general condition improves. The mistake made with ordinary vaccines is that too large doses are given, and they are repeated at too frequent intervals. If there is a definite result after injecting five million, you should not be in a hurry to give a second injection; do not give your second injection until a period of ten days or a fortnight has elapsed, and then do not give more than seven million; perhaps it is better to give another dose of five million. There is never any necessity, with an ordinary potent gonococcal vaccine, to go beyond fifteen or twenty million, and the subsequent injections should be increased very slowly, that is to say there should not be less than seven or ten days' interval between each.

What are the disadvantages of using ordinary gonococcal vaccine? There are two: (1) It produces a negative phase, during which the patient is worse for forty-eight hours after each injection. (2) There is a certain train of symptoms which occur after vaccine treatment, and sometimes before the treatment is started, viz. the patient will tell you that he has vague pains all over the body, which he calls muscular rheumatism, pains in the extensor and adductor muscles of the thigh, headache, pain in the small of the back, slight photophobia and sometimes conjunctivitis. This train of symptoms many of you must have noticed, and it is due to the endotoxines of the vaccine. You may divide vaccines into two parts—the bodies of the bacilli, and the endotoxines from within the bodies of the bacilli. The former have the effect of getting rid of the lesion, and the latter have the effect of causing toxic symptoms, which bother patients, and lead many medical men to think that vaccine treatment makes the condition worse rather than better. I tried to see if I could avoid these toxic effects by giving the injections intravenously. I did not like

to give the ordinary vaccine, because I did not know whether the bacillary bodies might have conglomerated or not, so I autolysed the vaccine. Gonococci autolyse quickly; at ordinary room temperature this occurs in twenty-four hours. I started off with intra-venous injections of three million in a quarter of a pint of saline solution. The negative phase after intra-venous injection is practically negligible if the dose given be small. If you give twenty to twenty-five million there is a negative phase afterwards, but it is not severe. Not only is there no negative phase after intra-venous injection of a small dose, but there are practically no aggravations of symptoms, the discharge is not increased, and the lesions rapidly improve, and are not made worse for the time being. The disadvantage of the intra-venous injections is that you require about ten before your patient is cured of his disease, and unfortunately with so many injections you cannot avoid the toxic symptoms which I described as occurring after the ordinary vaccine. Besredka, working at the Pasteur Institute in Paris, succeeded in other diseases in avoiding the negative phase by sensitising his vaccine.

I need not enter into an explanation of the theory here, but sensitisation of a vaccine is effected by charging it beforehand with a serum which already contains antibodies—in other words a vaccine which has been left in contact with an anti-gonococcic serum. Using the anti-gonococcic serum obtained from a horse gave unsatisfactory results, so I obtained a human anti-gonococcic serum by giving a patient with bad gonorrhœal arthritis intra-venous injections of vaccine and then withdrawing a requisite amount of serum. A vaccine sensitised with such a serum gives excellent results. There is no negative phase, and provided that the serum contains anti-endotoxines, no toxic symptoms follow. In the case of a sensitised vaccine of this sort you can give very much bigger doses with impunity, viz., twenty, fifty and a hundred million, on three successive days. There is no local reaction at all, no general reaction, nor a focal reaction, the symptoms are not increased in the least and they begin to disappear almost immediately, and there are no toxic symptoms produced. The stimulation of the antibodies which causes the lesions to disappear, and which follows the twenty, fifty and a hundred million, does not go on for an indefinite period—usually for not more than twenty-one days. In some cases the patient is cured ere this, but more often that is not the case so it is wiser about the fourteenth day to repeat the three doses in strengths of 200, 300 and 500 million; as before on three successive days. Even a third trio up to a 1000 million is sometimes advisable.

On prostato-urethritis sensitised vaccine has more action than any other kind of vaccine, therefore in every respect it is far and away the best kind of vaccine we have at present. With regard to treating cases of arthritis with vaccines there is one point which I must not omit to mention, namely, that if a joint has a great deal of fluid in it, however potent the vaccine may be, the fluid of that joint will not disappear after treatment with that alone.

So if the fluid in a joint has not disappeared or decreased within forty-eight hours after the three doses have been given, the joint must be tapped and a Martin's bandage applied as quickly as possible, owing to the rapidity with which such joints are wont to refill.

Now a word or two about the treatment of soft sores. I am, of course, assuming that you are familiar with the diagnosis of this condition. When a patient comes to you with a sore which you consider to be a soft sore, do not let him out of your sight for at least two months, because a number of these cases which come as soft sores are cases of mixed infection: they have syphilis at the same time as they are infected with Ducrey's bacillus, but owing to the incubation period of the former being much longer than the latter, the syphilitic symptoms do not show until that time has elapsed, namely three to six weeks, when the soft sore should have gone. You can always tell when a soft sore will become syphilitic, because the sore does not heal but begins to become hard in certain parts. If the soft sores are on the prepuce and you cannot get the foreskin back easily, the question often arises as to whether you should circumcise your patient. Now I want to insist upon this: never put an instrument near a soft sore; if you attempt anything with a knife, however clear of the sore you may be you will be certain to infect the whole wound, and the patient is worse at the end of your attempt than at the beginning. The bubo which results from a soft sore may come on after a

soft sore has healed. If you have a bubo in the groin which requires operating upon do not make a big incision, as is usually taught in general hospitals and is practised at the present day; if you make a big incision the wound will often take as long as six months to heal up. Never make more than a small opening with a bistoury, and having done that, squeeze the pus out, syri n gethe cavity with saline, and put a piece of gauze into the opening; this will enable your patient to continue his work, as the incision will heal in about ten days. Another disadvantage of making a big incision is because glands come into view and you are tempted to remove them, and the more glands you remove the more there are to remove. I have seen three cases where glands have been removed after operating for bubo in which the patient developed complete elephantiasis of one leg and elephantiasis of half his scrotum. So remember that in cases of soft sores, and buboes complicating them, operative treatment is practically never indicated, and that it is not even necessary to open a bubo unless that bubo is pointing.

Now I want to say a little about the treatment of syphilis. Far and away the most important point to remember is that the treatment of syphilis should be commenced the moment the sore is diagnosed. If you can treat a case of syphilis before the glands in the groin are enlarged or before secondary symptoms appear, it seems in the light of our present imperfect knowledge that a cure is certain; but once the secondary stage has commenced the word "cure" must be used with reserve. It is so often stated that you should never diagnose a sore as being syphilitic unless you have seen the *Spirochæta pallida*, but that is wrong, because many who are in the habit of seeing sores can diagnose a syphilitic sore by the look of it at once, and because there are syphilitic sores in which it is impossible to find the *Spirochæta pallida*. If a patient comes to you with a syphilitic sore, start your treatment at once, and if the sore is in a position from which it can be removed, it is best to remove it surgically. If you cannot remove it in this way, cauterise it, or rub in ordinary unguentum hydrarg. until all trace of the induration has vanished. Induration is a constant source of danger, because owing to the non-vascularity of the fibrous tissue the drugs used may not reach or kill all the organisms, with the result that a sore may break down later and give rise to a fresh general infection. If treatment is begun before the secondary symptoms are apparent, a Wassermann's reaction should be done before the first injection is given and again about forty-eight hours after, because you can therefrom form an opinion as to how much treatment will be required. The first Wassermann's reaction is not done for diagnosis, because the Wassermann test does not become positive until the disease is in the secondary stage, *i. e.* until about six weeks after the infection, but it is done as a control and to compare with the test done after the first injection, which in most cases becomes positive. Then so many injections of salvarsan should be given at weekly intervals until the Wassermann's reaction becomes negative, and a thorough course of mercury added thereto. Now here comes a very difficult point: How long should one keep a patient under mercurial treatment? It is a question which I cannot answer with absolute certainty, yet I can only tell you what I do myself, and warn you that it is quite empirical. I give the patient mercurial treatment for a year. We have no means at present which enable us to absolutely tell whether a patient is cured of his disease or not; because a patient gives a negative Wassermann's reaction is no certain indication that he is absolutely cured; and if he gives a positive Wassermann it is no certain indication that he is actively syphilitic though highly probable. One may argue that the Wassermann is positive because the patient has had syphilis, and no one can argue against that in the light of our present knowledge. The reasons which have compelled me to acknowledge this state of uncertainty have been produced by a discovery I have recently made, and which I will briefly refer to at the end of my lecture. Empirical as one's treatment must be until we know more about certain things, it is as well to have a routine, and mine is to aim at getting a negative Wassermann's reaction with salvarsan in the early stages of syphilis, and to follow the arsenic treatment up with mercury and iodides for a year. About four injections of salvarsan will be required in the primary stage, and in the secondary stage about seven. In some cases in the latent stage and in many in the tertiary it is impossible even with fifteen injections of salvarsan to obtain a negative Wassermann's reaction; in these cases my rule is merely to cure the symptoms.

Now I want to say one or two words about mercury. The form of mercury treatment which I give is nearly always intra-muscular injections of Captain Adams' grey oil: hydrarg. 20 parts, anhydrous lanoline 30 parts, chlorbutol 2 parts (all the foregoing by weight), liquid paraffin to 100 by measure. This grey oil is liquid at room temperature, therefore no heat is required to melt it, and 5 ♏ of the oil contain one grain of mercury; so the bulk is very small, and you can give from 5 ♏ to 15 ♏, that is three grains of mercury a week, without producing signs of mercurialism; and if you do not like to make the injection into the buttocks, a good place is into the scapular muscles.

I give three courses of eight weekly injections in a year, and after each course give iodides for three weeks; during the remaining five weeks no treatment is prescribed.

With regard to the question as to which is the most potent form of mercury, to calomel must be given the palm, but unfortunately its use is often accompanied by such great pain that in private practice it is not safe to use—that is to say, if you wish to keep your patients. If there is anything to interfere with the patient coming up for mercurial injections, for instance, if he has to go abroad, etc., you must give the mercury in some other form, either internally or in the form of mercury suppositories. The latter mode of administration has much in its favour, as gastro-enteritis is avoided, and likewise the depression which so commonly follows mercury when it is taken by the mouth. A mercurial suppository which is very useful is hydrarg. salicyl. gr. j. ol. theobrom. *q.s.*, one to be inserted every night. Mercurial inunctions are of course excellent, but only of use when they can be carried out by a trained rubber.

There is a very important stage of syphilis upon which a few words should be said, and that is the latent stage. Any number of you must have had cases who have had syphilis, and who come to you wishing to know whether they are cured of the disease or not. You can find no symptoms, and the Wassermann test is negative. You may perhaps tell the patient he need not bother, as the Wassermann reaction is negative, and that probably he is cured of his disease, but where marriage is concerned more caution should be exercised, and the patient should be advised to have a provocative injection of salvarsan. If the patient gives a negative Wassermann's reaction, having had syphilis, and you give him a provocative injection of "606" and he is not cured of the disease, he will give a positive reaction, which usually becomes positive forty-eight hours after you have given the injection. If not, it may become positive on the fifth or seventh day. Many of the cases in which the reaction becomes positive about the forty-eighth hour become negative again about the seventh day, and do not remain definitely positive until after a second injection of "606" has been given. If the patient wishes to be cured of his disease and to marry, you should advise him to have about five injections or more until the reaction becomes negative; but a cure should not be guaranteed. The provocative injection is valueless if the previous treatment, especially if it has been with salvarsan, has been recently administered—within six months.

However drastic treatment is, it is by no means always possible to obtain a negative Wassermann's reaction in tertiary syphilis, but you can tell your patient that recurrences are less frequent the further removed their appearance from the infection. For instance, if you have given the patient insufficient treatment in the primary or the secondary stage he may have a recurrence in three or four months; but if he has had insufficient treatment in the tertiary stage he may not have a recurrence for four or five years, and probably longer.

Consequently in the tertiary stage I give two injections of "606" and follow these up with mercury and iodides for a few months. I do not tell the patient that he will be cured of his disease, but I say the chances are that he will have no recurrence for many years, and it is quite likely that when he dies it will be from another disease.

I cannot pass by the treatment of syphilis without a short allusion to women who are infected with the disease during the child-bearing period. It is perfectly safe to give women who are pregnant "606" at any stage of their pregnancy. A woman who is pregnant and who is syphilitic always runs the chance of bearing syphilitic children, whether the father of her child has had syphilis or not. Therefore once a woman is syphilitic she can always give birth to syphilitic children, and the only way you can save her from that is to treat her throughout the course of each and every pregnancy. Give the patient as early as you can about four injections

of "606," and continue with mercury until the child is born. You will see many cases of women who have no signs of syphilis at all and give a negative Wassermann's reaction, but in spite of this give birth to syphilitic children; their first symptoms of syphilis often not appearing until after the menopause. The treatment of congenital syphilis with "606" is on the whole unsatisfactory, and is best prescribed when mercury and iodides fail to do what is required. The question as to how long one should continue treating a case of congenital syphilis cannot be satisfactorily answered; I certainly do not think it should be less than three or four years. In syphilis hereditaria tarda "606" is of very limited use, mercury being the only form of treatment which seems to do any good, but no stage of syphilis is more hopeless to treat.

Time will not permit me to make more than the briefest reference to nerve syphilis. For the sake of convenience we will divide the cases into three groups: (1) Cerebro-spinal syphilis, (2) tabes, and (3) general paralysis of the insane. Treatment of cerebro-spinal syphilis with "606" often yields remarkable results. In the case of tabes I think "606" had better be withheld and mercurial treatment given instead. I do not wish to give the impression that cases may not be benefited by salvarsan, as I have seen many such instances, but recurrences are almost invariable, and in many cases the symptoms are from the first no doubt aggravated. With regard to general paralysis of the insane I am sure "606" makes the patient a great deal worse. I have known patients with general paralysis of the insane who have had practically no symptoms except inequality of pupils, but who after one or two injections of "606" developed profound symptoms which resulted in death three or four months afterwards. Though the giving of "606" may temporarily improve such patients, I am certain that it eventually hastens their end. In all cases of nerve syphilis examination of the cerebro-spinal fluid should never be omitted, because it gives such a valuable clue as to the correct diagnosis. Clinically it is often difficult to diagnose a case of cerebro-spinal syphilis from tabes, but examination of the cerebro-spinal fluid will not infrequently help you out of the difficulty.

In conclusion I want to say a few words about some research work which I have been doing which may probably alter some of our present views of syphilis concerning both treatment and the current interpretation of the Wassermann's reaction. Hitherto you have learnt that syphilis is due to the *Spirochæta pallida*, and that everything syphilitic is due to that organism. If you think for a moment you will see that that cannot be the case, because if the *Spirochæta pallida* accounts for every syphilitic phenomenon, and you know that you cannot find the *Spirochæta pallida* in chancres, even after the first injection of "606," why do patients have recurrences? And why should you have to give more than one injection of that remedy? There must be stages in the cycle of the syphilitic organism other than the *Spirochæta pallida*. I have found other stages in the life-cycle of the syphilitic organism, and moreover shown that the treatment which we have at present does not kill them all, as some of the bodies which I shall briefly describe to you I have found in the lymphatic glands of patients who have had "606." So you see, gentlemen, that at present many of the questions concerning syphilis must still remain *sub judice*. Unfortunately we cannot definitely state how much treatment any case of syphilis requires, and neither can we feel absolutely justified in saying that a positive Wassermann reaction is indicative of active syphilis, or that a negative reaction means the cure of the disease, because we do not know what part the various phases of the syphilitic organism play in the production of symptoms or are responsible for the positive serum reaction.

I must draw your attention to another point which will show you that the *Spirochæta pallida* cannot be the sole cause of syphilis, because if the infection is brought about by the passage of spirochætæ from one person to another, you would expect to see spirochætæ in the act of division in every film you look at, but you never do. I do not think the *Spirochæta pallida* has need of division, as I take it to be a perfect phase in itself, and probably the adult male of the parasite of syphilis. I think it is most probable that the *Spirochæta pallida* is the male gamete of the male sexual cycle; in which case the *Spirochæta pallida* could scarcely be the infective agent of the syphilitic parasite. I think the infective agent of the syphilitic parasite is a small granule which I call the infective granule; being motile, it becomes

intra-cellular, and in the large mononuclears undergoes development, and becomes the female or the male part or develops asexually. The male part develops into a coil, and this coil then breaks up into irregular wavy bodies, from which the *Spirochæta pallida* ultimately is formed. The female body soon becomes extra-cellular, at which stage it appears to be made up of four or five bodies, which in time fuse, while by other methods of staining it appears as a round cell with a chromatic network inside. As a result of conjugation—an act which I have once witnessed—a zygote is formed which divides into two and subdivides into four, and so on until a cell is formed which contains numerous sporozoites or infective granules. Naturally these bodies vary in appearance according to the way they are examined. They are best studied *in vivo* stained with borax methylene-blue, then in fixed films stained with Giemsa, and finally in sections stained with pyronin and methyl green. I have also found similar bodies in the lymphatic glands of rats infected with *Trypanosoma Lewisi*.

Quite a general opinion prevails that the last word on syphilis has been said, but you will have noticed from what I have told you that most of the knotty points are still without solution, and until they have been freed of their yoke you must have an open mind of the whole subject.

December 30th, 1912.

Paryoxsymal Bradycardia.—The first case described by Laslett exhibited sinus arhythmia of a grade which is unusual at any age, and very rare beyond middle life. During the long auricular pauses multiple ventricular extra-systoles occurred. The patient suffered from attacks of giddiness, which, it is suggested, were due to cerebral anæmia induced by the long intermissions of the pulse. The longest intermission so far observed was two and a half seconds in duration. The arhythmia temporarily disappeared after the administration of atropin, and was, therefore, in all probability, vagal in origin. In the second case a true bradycardia of almost regular rhythm was found shortly after one of the cerebral attacks from which the patient suffered. It is suggested, though not proved, that the infrequent rate of the heart was due to increased inhibition.—*Journ. A.M.A.*, vol. lix, No. 23.

We have received from Mr. Henry Frowde and Messrs. Hodder & Stoughton a copy of 'Diseases of Women,' by T. G. Stevens, M.D., B.S., F.R.C.S. The author in his preface says this book was undertaken at the invitation of the Directors of the University of London Press, and it is the practical outcome of ten years' experience in teaching gynæcology to students. The lesson he learnt during that time is that gynæcology must be presented on a pathological basis, in the same manner as medicine and surgery, and that when presented in this manner the theoretical side, at least, of the diseases of women is no harder to acquire than that of the sister sciences. There were formerly many mysterious conditions in connection with the diseases of women, which are now surely explained by their true pathology, and in general it may be truly said that the great improvements which have been made in the treatment of pelvic disorders, are the practical result of general surgical principles applied on a sound pathological basis. Whilst striving to draw up a classification based on pathological processes, the author adopts a compromise between a purely pathological and an anatomico-clinical classification. The illustrations, which have mostly been drawn by Dr. Stevens, have been selected so as to illustrate as clearly as possible the various conditions described, without overcrowding the book with pictures, no doubt interesting, but not always illustrative. A certain number of photomicrographs have been used, unaltered by any attempt to transform them into diagrams because Dr. Stevens holds that the student should be encouraged to compare actual sections with photomicrographs, if the latter can be produced sufficiently well for the purpose. The difficulty is to produce them sufficiently well. The large photomicrographs have been produced by a method which the author believes has not been used before, namely, by direct photography upon a plate exposed in the rays of the epidiascope. He does not claim that his efforts in this direction are perfect, but they appear to him to be an improvement upon the older method, and further experience will probably enable other workers to make still more improvement. We have much pleasure in drawing the attention of our readers to this very useful work, and can assure the author that his teaching will be widely appreciated.

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A BRIEF REVIEW OF THE AFFECTIONS OF THE ŒSOPHAGUS.

A Lecture delivered at the Hospital for Consumption and Diseases of the Chest, Brompton,

By W. CECIL BOSANQUET, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

I am afraid that I am sadly out of the fashion in choosing for the subject of a lecture any theme but tuberculosis, which appears to monopolise the attention not only of the lecturers at this hospital, but of the whole country at the present day to the exclusion of other topics. My excuse must be that I wished to find a subject which had not already been practically talked to death, and also to call attention, from the point of view of examination of the chest, to an organ of which the existence may be overlooked and of which the affections may therefore give rise to errors in diagnosis. Anatomically the œsophagus lies in relation to the trachea and pericardium anteriorly, while the aorta and recurrent laryngeal nerve are near neighbours, and the lungs and pleuræ come in contact with it on either side. We must remember that it has a short course in the neck and also in the abdomen, though these portions do not immediately concern us to-day. The veins of the œsophagus join the inferior thyroid, azygos and gastric veins, while its lymphatics drain into the cervical, bronchial and mediastinal glands. In structure it resembles the rest of the alimentary canal, being a muscular tube lined by mucous membrane, but the latter is covered with stratified epithelium. The gullet exhibits distinct peristaltic movements. It seems to possess some sensation of heat and cold, while painful sensations may also arise from it, though, as in the case of gastric ulcers and similar lesions of the lower alimentary

canal, the exact mode of their production may be doubtful.

Affections of the œsophagus are naturally accompanied by some disorder of the act of swallowing, and our investigation of a case of difficulty in swallowing should naturally begin by making the sufferer attempt to swallow something in our presence. As in some cases solids give rise to most difficulty, *i. e.* when there is organic obstruction; in others liquids, for example, when the trouble lies in the innervation of the tube, both kinds of food should be used for the test. In days gone by, the next procedure was to pass a bougie down the gullet, and with suitable precautions against doing harm—*e. g.* by carefully ascertaining the improbability of an aneurysm being the cause of dysphagia—this method may still be pursued, but it is preferable, if the means are available, to have the condition of the œsophagus determined by examination with the X-rays, for which purpose either a cachet containing bismuth may be used to represent solid food, or a suspension of a bismuth salt as a fluid test-draught. Finally, it is now possible to examine the œsophagus by sight, by means of the endoscope or œsophagoscope, a mode in which some of our laryngologists have acquired notable skill. It is well, however, to bear in mind when we read the enthusiastic accounts of this instrument, that its use even with the aid of cocaine is distressing to patients, and that for accurate observations even by an expert, a general anæsthetic is needed. It is a method only to be applied in cases of real necessity—conditions of gravity and of doubtful nature, or as a guide to operative treatment of conditions within the gullet.

Coming now to a consideration of the affections of the œsophagus, we may first consider the minor affections of a *functional* nature to which it is liable. These are spasm and dilatation, singly or in conjunction. Spasm of the œsophagus is said to occur either at the upper or the lower extremity of the tube. The existence of anything resembling a sphincter at the cardiac end has been denied by Hill, but the occurrence of some degree—even a marked degree—of spasm or contracture at this point is fairly well established. Minor degrees of the affection involving the upper extremity of the œsophagus are not uncommon in nervous individuals, if one may judge from the number of cases one sees in which the complaint is made that food seems frequently to stick in the throat and needs a special effort to pass it down the gullet. Some of these may be instances rather of pharyngismus than of œsophagismus. Thus I saw a case in which a neurotic lady of middle age complained of difficulty in swallowing soup, the mouthfuls of liquid tending to return into the mouth or even to be expelled from it: she experienced a choking sensation in trying to swallow and made curious spasmodic kicking movements of her feet at such times, the affection being worse if she had any tonsillar trouble at the time. The disturbance seemed to be an exaggerated and inco-ordinate reflex action.

Severe cases usually involve the lower part of the œsophagus and may lead to dilatation of the tube above the stricture. Thus, a medical man complained all his life of difficulty in swallowing, and of feeling that the food taken was in danger of being shot out from his mouth, the trouble being worse at times than others. He suffered from foul breath and "indigestion." He was subsequently found to have a dilated œsophagus.

Considerable difference of opinion seems to exist as to whether in these cases of œsophageal dilatation there is always a state of spasm or not, many authors maintaining that the condition is one of primary dilatation owing to atony of the wall of the gullet. Probably both conditions occur, differentiation being possible by passage of a sound. If the instrument passes easily into the stomach there is no spasm present, this being indicated by the way in which the muscular wall of the tube grips the sound and impedes its passage. In some instances hypertrophy of the muscular wall has been found above the seat of spasm, sufficiently proving its real existence. In these neurotic cases the difficulty in swallowing may be greater for liquids than for solids or the reverse. The trouble may start quite early in life. Umber relates that in one instance it appeared in both father and son; usually women are the chief sufferers. The symptoms are difficulty in swallowing, with sensation of food "sticking" in the chest, foul breath and eructations of fluid, and in severe cases weakness and emaciation. A tube passed into the dilated gullet will bring up alkaline material. Examination with the X rays after a draught of bismuth suspension will reveal the dilated condition of the œsophagus.

A curious case is recorded by May. A man,

æt. 55 years, a hasty eater with bad teeth, suffered from increasing difficulty in swallowing. His food seemed to stick about the end of the sternum. He vomited occasionally, and could cause his food to run out of his mouth by merely bending down. Sounds were passed with difficulty, thinner and thinner instruments having to be used. Examination of the chest showed bronchial breathing over the middle and lower lobes of the right lung and a systolic murmur at the apex of the heart. These physical signs disappeared when the dilated sac formed by the œsophagus was emptied. We have, therefore, to realise that affections of the œsophagus may give rise directly to physical signs suggestive of disease of the lungs and heart, though I do not wish to suggest that the next case of bronchial breathing over the base of the right lung which you encounter is to be diagnosed as due to œsophageal dilatation, in default of special evidence pointing thereto.

With regard to treatment of these conditions, the mild cases of spasm are best let alone, any undue notice taken of these neurotic troubles tending to concentrate the patient's attention on them and to aggravate the symptoms. In cases of greater severity patients often find out for themselves ways of overcoming the difficulties in swallowing, as by taking a drink of water or of some alcoholic beverage to help the food down. If the trouble is severe it may be necessary to pass a bougie from time to time, or even daily for some weeks.

Before leaving the subject of these conditions of spasm and dilatation of the œsophagus, it is necessary to point out that the latter affection is not always due to mere neurosis, whatever that may mean, but that cases have been described in which a paralytic condition of the tube has been caused by degeneration of the vagi after diphtheria, and that it may also exist in bulbar paralysis in which these nerves suffer. A primary œsophagitis has also been assigned as the cause of dilatation. Spasm of the upper end of the gullet may occur in cases of cancer at the lower extremity (Hill), and even in disease of the stomach (ulcer, Buchanan), or of the liver (Dundas Grant).

Coming to *organic* affections of the œsophagus, we have first to recognise that in rare instances *congenital malformations* are encountered, in the form of stenosis or actual atresia, the natural process whereby the œsophagus is divided off from the gullet going too far, and leading to obliteration of the lumen of the latter at the level of the cricoid cartilage. Small cysts lined by epithelium may be found on its walls, possibly representing in some cases remains of branchial clefts.

Rupture of the tube may take place as the result of vomiting, especially in alcoholic subjects; and a curious case is recorded by Roy, in which the gastric contents escaped into the pleural cavity and were blown out in the form of a fine spray with every expiratory effort, doubtless owing to the attendant rise in intra-thoracic pressure forcing them up to the top of the gullet and over the opening of the glottis. Rupture may be caused by attempts at dilating a stricture, as in a case related by Whipham and Fagge, in which this accident occurred in a child the subject of congenital stricture, the rupture causing right pneumothorax. Subcutaneous emphysema may also be found in these cases.

Foreign bodies from time to time become impacted in the œsophagus, especially tooth-plates and fragments of bone. Pain is generally felt, assigned to some point behind the sternum, but it is interesting to note that in many cases it is found that the foreign body escapes unnoticed, and that the pain is due to the presence of a local ulcer which it originally caused. Thus Ewald notes an instance in which a plate with five teeth passed through the whole alimentary canal and was never found, a sore point in the œsophagus, probably due to an ulcer, being the only symptom of trouble. Such an ulcer may prove fatal, as in a case recorded by Wallis, in which a woman swallowed a fragment of chicken-bone; subsequently hæmorrhage occurred and death ensued, a perforation of the aorta being found at the necropsy.

The interesting subject of *diverticula* of the œsophagus does not very directly come into relation with affections of the chest, so that I will not deal with it at length. Diverticula have been divided into (1) pulsion diverticula, in which the mucous membrane is forced through a gap in the muscular coat; these occur at the level of the cricoid cartilage and may originate in some congenital weakness of the muscular wall at this point; and (2) traction diverticula, in which some outside part becomes adherent to the œsophagus, and by subsequent contraction pulls upon its wall and causes a local bulging. The part most often at

fault in these latter cases is a tuberculous gland, but it is said that inflammation may start in the œsophagus itself and spread outwards, giving rise to adhesions elsewhere, which may result in traction and pouching. These traction diverticula, which occur in the anterior wall of the œsophagus and lie deep in the chest, do not give rise to symptoms of dysphagia. Practically they are found as surprises at post-mortem examination. On the other hand, pulsion diverticula, which are situated in the neck and involve the posterior wall of the gullet, betray their presence by definite signs. Difficulties in swallowing are noted early, and it is found that food taken, especially fluids or semi-fluid matters, is regurgitated. The patient may speak of it as vomiting, but the reaction of the material brought up is alkaline, showing that it is not derived from the stomach. Sooner or later a swelling is noticed in the neck, and the patient may discover for himself that he can empty the sac by pressure with his hand. Pressure of the full sac on the sympathetic may give rise to narrowing of the pupil and palpebral fissure on the corresponding side. Even the radial pulse may be altered in some instances by pressure on the subclavian artery at the root of the neck. If decomposition takes place in the contents of the sac, the patient's breath may be very offensive. Diagnosis is easy when the external swelling has occurred, the escape of food-materials on pressure upon the sac proving that the condition is not one of retro-pharyngeal abscess. Administration of bismuth capsules or emulsion and examination with the X rays will also reveal the nature of the lesion. Treatment is by excision of the sac and suture of the orifice leading into the gullet. It is not an especially difficult operation, and the results are very satisfactory.

We now come to the inflammatory affections of the œsophagus, which may be superficial, involving the mucous membrane only, or extend deeply into the wall of the tube, or even through it to the parts outside. A close resemblance exists, as we shall see, between the affections of the œsophagus and those of the stomach, and the two organs may suffer together.

Perhaps the simplest condition, if it can be properly called inflammation is *herpes* of the œsophagus. The eruption may be present in the mouth at the same time, thus facilitating the diagnosis. According to Holub the symptoms are attacks of cutting pain between the shoulders, and in the chest and epigastrium, lasting for a few minutes, and followed by difficulty in swallowing. The lesions have been seen by means of the œsophagoscope. Treatment would consist in temporary abstention from solid food, and if necessary the use of cocaine lozenges or ice to suck. Patches of thrush may develop in the œsophagus in debilitated subjects—the usual fungus, *Oidium lactis* being sometimes associated with other organisms (oöspora, Dunan).

A condition of painful *fissure* of the mouth of the œsophagus was described by Bronner at the recent meeting of the British Medical Association. It accompanies a condition of dry catarrh of the pharynx, and affects principally women between 30 and 50 years of age, giving rise to pain in swallowing. The condition may last for years, and lead to some degree of emaciation. It is easily cured by passage of a bougie, which relieves the spasm set up.

Little is known of simple catarrhal inflammation of the œsophagus (*œsophagitis*), but judging from the comparative frequency of cases in which slight pain in swallowing is complained of, without obvious cause, and of a transitory nature, it is not uncommon, as might indeed be expected in a tube which, like the gullet, is exposed to frequent irritation from hot or stimulating foods. In the case of those examples of dilatation of the œsophagus which are attributed to œsophagitis, it is at least as likely that the inflammation was secondary and due to irritation of retained decomposing food as that it was a primary cause of the trouble. The inflammation which follows the ingestion of violent irritants, such as acids and alkalies, is well known, and in bad cases destruction of much of the œsophageal wall may ensue, with subsequent scarring and stricture. In cases of acute injury Janowsky recommends the use of suprarenal extract by the month; ice and cocaine may also help, and feeding must be carried out *per rectum*.

A peculiar form of inflammation occurring between the epithelial and subepithelial layers is described by Stern, and named by him *œsophagitis dissecans*. He records a case occurring in an alcoholic subject, symptoms being nausea and vomiting, with pain in taking solid food and some fever. Subsequently the patient brought up a cast consisting of the mucous membrane of the œsophagus,

13 cm. long, and finally recovery ensued. *Phlegmonous inflammation* of the œsophagus has been seen by Pfister accompanying a similar condition in the stomach. The gullet was found infiltrated with pus and there was suppuration in the posterior mediastinum.

Ulceration of the œsophagus may be due to a variety of causes. The simplest is the so-called decubital ulcer, due to pressure of the gullet-wall against some hard stucture in the neighbourhood, as for example, against the cricoid cartilage or against a bony projection from the vertebral column. These ulcers do not seem to give rise to symptoms. Ulcers due to lodgment of foreign bodies have already been alluded to. Mazzotti reports five fatal cases of what he terms "ulcerative œsophagitis," the symptoms being pain in swallowing and pain in the abdomen, fever and bloody stools, the condition resembling gastric disease. The ulceration of the gullet is usually associated with ulceration of the colon, to which presumably are due the abdominal symptoms. The patients affected are between fifty and seventy years of age, and the phenomena suggest an infective cause. Ulceration of the gullet may occur in diphtheria, scarlet fever and enteric fever, and may be followed by cicatricial stricture.

Peptic ulcers, of the same nature as the ordinary gastric ulcer, may be met with in the œsophagus. The symptoms are pain, felt immediately after food is taken, either at the lower end of the sternum or in the back, and tenderness when a bougie is passed. These ulcers, being associated with the action of acid gastric juice, necessarily occur at the lower end of the gullet, and they would seem to constitute a dangerous malady, as Kappis, who collected fifteen cases, found that ten of these ended fatally, while five were found accidentally at post-mortem examination. The occurrence of scars and strictures at the lower part of the tube suggests that such ulcers may be more common than is usually recognised, and that the percentage of fatal cases is not so high as the figures just given would lead us to think. The principal causes of death are hæmorrhage and perforation of the ulcer. In one case the aorta was penetrated, in another pneumo-thorax occurred, and in a third the perforation was below the diaphragm, and peritonitis carried off the patient. Kappis relates the history of a case which must be almost unique in view of the variety of operative measures undertaken. A woman, æt. 33 years, who had had hæmatemesis and symptoms of gastric trouble at the age of thirteen, complained of difficulty in swallowing solid food. Examination with the œsophagoscope revealed stenosis of the gullet, and gastrostomy was performed. Subsequently vomiting ensued, showing that the stenosis was merely due to spasm—an example that even the most recently invented instrument may not always prove infallible—and a gastro-enterostomy was done, but also proved unsuccessful in relieving symptoms. A second operation of this nature was, therefore, carried out without better success, and finally it was found that there was a condition of hour-glass stomach, and a jejunostomy was effected. "Last of all the woman died also," and at the necropsy there were found a condition of hour-glass stomach, due to a cicatrised gastric ulcer, and also a healed ulcer of the œsophagus. A good many morals might be drawn from this instructive case, but I will only use it to illustrate the frequency with which lesions of the œsophagus coincide with others in a different part of the alimentary canal.

The treatment of a peptic ulcer of the œsophagus is similar to that of a gastric ulcer—rest in bed, abstinence from solid food, and if symptoms are severe, rectal feeding. It is interesting to speculate whether some of the cases in which hæmatemesis has occurred and no ulcer has been found in the stomach at operation may really have been instances of œsophageal lesion. The diagnosis between the two maladies is not by any means easy, though the pain in the gastric affection is usually rather lower down than that experienced when the gullet is effected; the latter is often retro-sternal, and is felt more immediately after the attempt to swallow food.

Tuberculosis may occur in the œsophagus in rare instances and usually takes the form of ulceration. Great difference of opinion seems to exist as to the frequency of such infection, Louis stating that the œsophagus is affected in 5 per cent. of all cases of pulmonary tuberculosis, while Grousdeff puts the proportion at 1 in 3700 cases. Other writers give intermediate figures. We may be content to recognise that the gullet is an uncommon seat of tuberculosis. Primary lesions due to the tubercle bacillus are probably never

found in it, and almost all recorded instances of secondary affection have been in cases of chronic pulmonary disease. Staehelin-Burckhardt, however, saw one instance in a girl, æt. 14 years, the subject of generalised miliary tuberculosis, and the existence of phthisis is not actually noted in one of Guisez' cases. Pain is generally very severe and may lead to total refusal of food, though it is said that sometimes these ulcers may be painless. Salivation may be a troublesome symptom as in other grave œsophageal affections. The œsophagus is probably invaded by the blood stream or lymphatic channels, not by direct inoculation with swallowed sputum, but the latter possibility cannot be quite excluded. Occasionally it is invaded from without by extension of disease from a tuberculous gland or from a focus of tuberculosis in one of the vertebral bodies. Pressure from such a gland may give rise to difficulty in swallowing without actual extension through the wall of the tube. A sclerosis of the gullet-wall due to tuberculosis has been described by Schrotter. If ulcers can be seen with the œsophagoscope, they should be painted with lactic acid, which may lead to their disappearance in favourable instances.

Syphilis may from time to time be met with in the œsophagus, and cases of unexplained stricture have been assigned to its influence. A mucous patch was actually seen in the œsophagus by Stark in a woman, æt. 27 years, and instances of ulceration and stenosis are recorded by Guisez and Abrand, who also saw an instance of definite gummatous formation in the gullet of a man, æt. 50 years.

We now come to the most important diseases of the œsophagus, simple stricture and malignant disease. *Fibrous stricture* may follow any condition which causes deep destruction of the wall of the tube, and the ingestion of corrosive fluids is a frequent agency. Ross records a case in a child, æt. 2 years, as a result of swallowing scalding water, and I recollect an instance in a girl of about five and twenty, who had taken hydrochloric acid with suicidal intent and recovered from the immediate injuries produced. Traumatic strictures of this nature may be followed by tuberculosis of the gullet, as in cases recorded by Rochner and Staehelin-Burckhardt. Ulcers of any kind may be followed by sufficient scarring to give rise to stricture: thus instances are recorded following diphtheria and scarlet fever (Jungnickel, Boas). Guisez records a series of cases in which the narrowing of the lumen of the œsophagus by cicatricial tissue could not be assigned to any antecedent disease, and which he calls "spontaneous cicatricial stenosis." The term is not very happy, as a growth of scar-tissue is unlikely to occur without preceding inflammation. He suggests, indeed, an œsophagitis as the real cause. The condition occurred in elderly persons, three women and two men, who gave no history of injury and were presumably free from syphilitic taint. Cases very closely resembling fibrous stricture are those described as due to hypertrophy of the muscle of the gullet-wall and to diffuse myoma, the latter condition being said in some instances to involve a considerable length of the tube.

Diagnosis of simple stricture has to be made from compression of the œsophagus from without and from cancer. The characteristic symptom is gradually increasing dysphagia, for solid food first and later even for liquids. It is important to eliminate the existence of external pressure due to an aneurysm before instruments are passed down the gullet, for fear of such a fatal catastrophy as perforation of the sac. Examination with the X-rays, which may be used to localise the position of the stricture—capsules of some bismuth salt being administered and the point at which they are arrested observed with the screen—will reveal the existence of any aneurysm large enough to cause dysphagia. It will also indicate with fair certainty the presence of a mediastinal growth or dermoid cyst or even of enlarged glands, which last may be either tuberculous or lymphadenomatous, or more rarely cancerous. In a young person a history of preceding injury will point to the likelihood of simple stricture, but in an older person examination with the œsophagoscope is advisable to distinguish between simple and malignant stricture, with a view to vigorous treatment of the former condition if it is discovered.

This is carried out by passage of bougies of increasing size, much as in the case of a urethral stricture. Progress must be made with caution, as rupture of the tube may occur and is a dangerous accident. Injections of thiosinamine or fibrolysin are said to be of assistance in softening the fibrous tissue. Electrolysis is recommended by Züber-

buhler. If the stricture is impermeable from above even by fine bougies, it is possible in some cases to perform retrograde bougieing from the lower side of the stricture through a gastrostomy wound. Ewald, who points out that bougieing should not be carried out in cases due to caustics until acute symptoms such as suppuration have ceased, mentions a case in which resection of a fibrous scar was successfully performed.

New growths of the œsophagus comprise sarcoma, epithelioma and spheroidal-celled carcinoma, epithelioma being considerably the most common.

Sarcoma occurs either as a polypoid tumour projecting from the wall of the tube or as an infiltrating growth. Patients are usually over forty years of age, and males predominate over females in a ratio of 3 to 1. The lower third of the gullet is most often affected, and actual stenosis is not so often encountered as in cases of cancer proper. Dysphagia is complained of, without obvious obstruction recognisable on passage of a bougie, and there is often a considerable degree of pain. The course of the disease is rapid, with asthenia and emaciation. Metastatic deposits are said to occur in the bones. A mixed form of tumour consisting of carcinoma and sarcoma combined has been found in the œsophagus. It is interesting to remember that mixed tumours have been found to develop in mice inoculated with cancer derived from another mouse.

Cancer develops in the œsophagus at three main seats of election, the upper extremity, the middle where the tube is crossed by the left bronchus, and the lower end where it joins the stomach. In this last situation the growth may spread into the stomach or cardiac cancer may spread into the gullet. Diagnosis of the exact point of origin may be difficult, and statistics as to the prevalence of this tumour may therefore be untrustworthy. Recent figures seem to show that the middle of the tube is the part most often diseased. Cooper, for example, states that the part behind the bifurcation of the trachea is affected in 40 per cent. of the cases, the lower end in 30 per cent., and the upper extremity in only 10 per cent. Looking through the cases which have come to necropsy at Charing Cross Hospital I find an almost exactly similar ratio, the majority having been situated in the central part of the gullet (40 per cent.), 30 per cent. at the lower end, and 20 per cent at the upper.

The three points mentioned are places at which the œsophagus is somewhat narrowed physiologically, and it is supposed that irritation is most likely to be caused at these points, and that the frequency of cancer there is thus explained. In a case reported by Wolf the disease originated at a point compressed by a bony growth due to spondylitis deformans. As in the stomach, cancer may be found in the gullet starting in an old ulcer; thus, Bell and Macrae record a case of this sequence in a woman, æt. 32 years, an unusually early age, the ulcer dating back ten years and being cicatrised. Cancer of the œsophagus may co-exist with malignant disease in some other part of the alimentary canal; it is noteworthy that it is not very uncommon for two foci of the malady to be found simultaneously in this tract, the structure of the two tumours not being necessarily the same. Sometimes a second focus of disease is formed in the gullet itself when the upper or lower extremity is primarily involved, owing to infection of glands lying at the bifurcation of the trachea, from which the growth extends into the middle part of the œsophagus. In comparatively rare instances intermediate portions of the tube are the seat of cancer, not the three seats mentioned above, and not very infrequently so considerable an extent of the wall is involved that it is difficult to ascertain at what point the tumour originated.

Secondary deposits are often absent in malignant disease of the œsophagus, but when they are found they may be situated in a variety of different places. The neighbouring glands are most commonly affected, and probably microscopical investigation would reveal foci of cancer-cells within them in cases where no disease was visible to the naked eye. The glands above the clavicles may in some instances become enlarged and help in the diagnosis of the disease; sometimes, on the other hand, they may be affected so early and so extensively as to lead to error, as in a case of cancer of the upper extremity of the gullet in which the swelling of the neck led to a diagnosis of cancer of the thyroid body. If the glands break down rapidly they may be diagnosed as an abscess in the neck without any suspicion arising that a malignant growth is at the bottom of the trouble.

Pressure due to enlarged glands may manifest itself by paralysis of one or other recurrent laryngeal nerve. I remember a case of this kind

which I saw many years ago at Charing Cross Hospital. A man admitted under the care of Dr. Abercrombie had suffered over a year previously from such paralysis without any signs pointing to œsophageal trouble. He came into hospital at last with dysphagia, and the total duration of his illness was two years, a very long course for this affection. Cailliau records a case in which there was not only paralysis of the left recurrent laryngeal nerve but also an area of dulness below the left clavicle, extending downwards so as to be continuous with the cardiac dulness, and enlargement of the veins on the front of the chest, thus very closely simulating the conditions produced by an aortic aneurysm. Finally, dysphagia appeared and troublesome salivation, enabling a diagnosis to be made. In this instance the pericardium was finally invaded by the growth, and secondary nodules were found in the liver and pancreas.

Secondary growths in the liver are not uncommon, and occasionally this organ may be so extensively affected as to form the most striking feature in the clinical picture. This cause of error is more frequently encountered in cases of gastric cancer. It is well to remember that primary cancer of the liver is uncommon, though not so rare as used to be maintained, and carcinomatous enlargement of this organ should always suggest a primary growth in some part of the alimentary tract. The liver is more likely to be involved in cases in which the lower end of the gullet is the seat of cancer than when the disease is seated higher up. Similarly, sepsis from an ulcerating growth has been known to spread to the liver along the lower œsophageal and gastric veins and to set up suppurative pylephlebitis (Edwards). The distribution of secondary deposits in œsophageal cancer is often very curious; thus I find one case in which the supra-renal bodies were both affected, no other organ suffering; another in which both kidneys suffered, without other organs being visibly involved.

Other complications of the disease are caused by direct extension of the growth to neighbouring parts. I have found only two cases in which the aorta was perforated by an ulcerating cancer, one being recorded by Davidsohn, the other occurring at Charing Cross Hospital in a patient under the care of Mr. Boyd, the disease being situated in the lower part of the tube, and the point of perforation being found just above the diaphragm. The pericardium and even the walls of the heart are sometimes infiltrated. The trachea and lungs are, however, the organs most often affected. The trachea suffers whether the growth is situated at the upper end or the middle of the œsophagus, and the symptoms produced may be very distressing, the patient being seized with a paroxysm of coughing and suffocation on any attempt to swallow, or finding this action only possible in some special posture, as when lying on one side or on his back. Death in these cases is usually brought about by septic broncho-pneumonia. Extension to the lung generally results in the formation of a ragged gangrenous cavity, and the patient suffers from hectic fever and fœtid expectoration. If the pleural cavity is opened up a pleural effusion will probably be formed on the affected side, which may be purulent and stinking; pneumothorax may also ensue.

Cancer of the œsophagus in typical cases is one of the easiest diseases to recognise: the gradually increasing dysphagia in an elderly patient, the difficulty affecting first the taking of solids, then that of fluids also, combined with emaciation, weakness and cachexia, form together a characteristic picture. Salivation is also frequently met with, and large quantities of mucous frothy matter may be brought up. In not a few instances, however, the disease is curiously latent. No complaint may be made of difficulty in swallowing, and all that may be noticed is increasing weakness, emaciation and anæmia, which may end in fatal syncope or coma without the occurrence of any œsophageal symptoms. In this latency the malady closely resembles cancer of the stomach. In other cases the symptoms due to involvement of the lungs predominate and may be quite misleading. I well remember one such instance which I saw when I was house-physician to Dr. Green. A middle-aged man was admitted to hospital suffering from cough, offensive sputum and weakness, with an irregular, intermittent pyrexia. Examination revealed dulness and coarse bubbling râles at the base of the right lung, and a diagnosis of bronchiectasis was not unnaturally made; this appeared to be confirmed when the patient subsequently suffered from attacks of hæmoptysis. There was never any complaint of difficulty in swallowing, but at the necropsy there was found a condition of gangrene of the lung caused by extension of an ulcerating cancer of the œsophagus. With regard to the treatment of cancer of the œsophagus there is little to be said that is at all encouraging. Removal of the growth, which at present remains the only effectual way of treating malignant disease, is seldom if ever feasible, though

Arbuthnot Lane records a case in which he removed a tumour of the upper end of the œsophagus by operating through the neck and turning in a flap of skin to form a new section of the gullet in place of the part removed. He regards the results obtained as encouraging for further trial of the procedure, though as the patient could not swallow through the newly formed channel, and finally died of recurrence of the growth, it does not seem to afford grounds for great enthusiasm. The only other position in which removal would be possible is at the extreme lower part of the œsophagus where it becomes an abdominal organ. Apart from such removal the only method of directly attacking the tumour is by application of radium salts, and this has been attempted by several surgeons, the substance being enclosed in a small tube and passed down the gullet until it rests within the ring of growth. Some relief of pain and spasm is said to have been obtained by this means, and it is possible that disintegration of parts of the tumour thus produced might lead to temporary improvement in the power of swallowing.

Palliative measures consist in the passage and maintenance in the œsophagus of a tube which passes through the point of stricture and enables fluid food to be swallowed; the passage of bougies to dilate the stricture; and the performance of gastrostomy, to enable food to be taken by another route. The first may be of use in tumours of the upper part of the gullet, care being taken to fasten the tube securely to the teeth, or elsewhere, so that it may not slip into the stomach. Passage of bougies through a canal, the walls of which are lined and penetrated by soft vascular new growth, is a dangerous procedure, and should not be attempted except in case of some special need, which can hardly arise when the services of a skilled surgeon are available. Gastrostomy theoretically affords a means of enabling a patient to take food without the need of a free passage through the œsophagus, and it has been advised to operate early in these cases of cancer, before the patient is weakened by lack of food, and thus placed in direct jeopardy from the shock of the operation. I do not find that my surgical colleagues are now so ready to resort to this procedure as they seemed to be a few years ago, and it would appear that all the theoretical advantages of gastrostomy are not actually realised. At all events, patients seldom live long after the operation, so far as my experience goes; and Ewald, who has great experience of gastric surgery, holds that the operation is not of much use. So long as a patient can swallow milk and other fluids in adequate amounts it is best to leave matters alone, supplementing the quantity of nourishment taken by means of nutrient enemas if it seems desirable. If swallowing becomes impossible, and starvation is threatened, then gastrostomy may be performed as a last resource.

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January 6th, 1913.

LECTURES ON NEUROLOGY.

Delivered to Post-Graduates at St. Bartholomew's Hospital.

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LECTURE III.

SENSORY DISTURBANCES.

GENTLEMEN,—I want to talk this afternoon about the sensory system, and I wish to draw attention to certain points in connection with the varieties of sensory disturbance which are met with in nervous disease. We will first of all consider the peripheral sensory neuron, that is to say, the afferent path from its extremity to its ending in the spinal cord. You can divide the varieties of afferent impulses which pass along this peripheral neuron into those which underlie cutaneous and deep sensibility. Cutaneous sensibility, you will remember, includes tactile, thermal (hot and cold), and painful sensations; and under tactile sensibility you include tactile localisation and tactile discrimination; that is to say, the power of distinguishing two points of the compass as two points, and the power of recognising different textures when the eyes are blindfolded; for instance, the difference between satin, silk, calico, and so on. Deep sensibility includes muscle sense, afferent impulses from tendon and joint sense. As a result of muscle and joint sense we appreciate the sense of passive movement, the sense of position, and so on. And it is owing to the deep sensibility remaining intact that co-ordination of muscular movement is possible. If a patient's deep sensibility is destroyed, his movements become incoordinate, and also equilibrium is disturbed.

As an example of injury to the peripheral neuron let us consider what happens from the point of view of sensory disturbance if the median nerve is divided at the wrist. Unless you cut the tendons at the wrist as well as the nerve you will find no alteration in the deep sensibility. You can divide the median nerve, and you can take a pencil or pen and press on the hand, and you will find everywhere this pressure is appreciated. You are stimulating those fibres which carry impulses of deep sensibility. So if you only test the hand in this way after median nerve division, you might be led to suppose that sensation is very little if at all affected. If the tendons, as well as the nerves, are cut, there is a different state of affairs, and deep sensibility will be lost. The reason for this is that the nerves which carry deep sensibility to the hand leave the median fairly high up in the arm, running with the tendons at the wrist to be distributed to the muscle. Supposing deep sensibility remains practically intact, what alterations do you find in cutaneous sensibility? I can best illustrate that for you by means of this lantern-slide, which illustrates the loss in cutaneous sensation following division of the median nerve. There is a dark outline here which represents in each case the area within which sensation is altered, and you will notice that it corresponds to the ordinary anatomical distribution of the nerve, thumb, and two and a half fingers. Within that area there is a part which is shaded black, and a part between the black area and the dark outline which is white. The black shaded area corresponds to the part in which there is total loss of cutaneous sensation, but in the white area sensation was altered, but was not entirely lost. That area varies in different cases. There are two varieties of cutaneous sensation which have been described by Head; one he calls protopathic, and the other epicritic. In the dark shaded area both protopathic and epicritic forms of sensation are lost. In the white area the epicritic sensation is lost, but the protopathic sensation remains. It is characteristic of injury to a peripheral nerve, that there is an area of complete loss of sensibility to all forms of cutaneous stimuli, and an area where epicritic sensation is lost, but protopathic sensation remains. One must explain what is meant by epicritic and protopathic. Epicritic means the power of appreciating light touches, such as touch of cotton-wool, and localising those touches. In addition to that the power of discriminating between small differences of temperature, say the power of recognising the difference between 20° and 25° C. That power depends on the epicritic sensation. By protopathic sensation one means the recognition of pain, and also the power of distinguishing between differences of temperature of great degree, such as between 0° and 50° C. In this white area the patient can distinguish

between 0° and 50° C., but if you were to test his sensation with water at 25° C. and water at 30° C. the patient would not be able to distinguish between them. This protopathic and epicritic sensation is the essential characteristic of the peripheral nerve. When we come to the spinal cord and find injuries to that, all distinction between protopathic and epicritic sensation is lost: these are purely a function of the peripheral nerve. Within the cord sensations are linked up in a different manner. There is another point in connection with this peripheral neuron that I must mention, and it is that the distribution of the sensory loss on the body varies according to whether it is a peripheral nerve which is injured, or whether it is one of the posterior nerve-roots which is affected. The closest correspondence between these two occurs in the intercostal nerves, where the nerve roots and peripheral nerves correspond fairly closely together. But in the brachial plexus and the lumbo-sacral plexus there is a difference between the root supply of the skin of the limb and the peripheral nerve supply. This diagram illustrates the segmental distribution of the posterior roots. I think most people are familiar with the sensory peripheral nerve distributions, but they are not so familiar with the segmental arrangement, though it is comparatively simple to remember. The fifth nerve, of course, supplies the face, and the first cervical segment apparently has no cutaneous sensory distribution at all. The second, third and fourth cervical segments form a kind of girdle, taking in the neck and top part of the shoulders. If you hold out the arm at right angles to the body, the fifth segment comes along the upper margin of arm and shoulder; then comes the sixth, including the thumb and index finger; the seventh is a narrow strip, which takes in the middle finger. The eighth takes in the ulnar half of the hand, the first dorsal, the lower part of the arm and elbow. The second and third dorsal take in the lower part of upper arm and axilla, and then you come to the fourth dorsal again on the chest. So about on a level with the angle of Ludwig on the chest is a gap where you pass from the fourth dorsal to the fourth cervical segments. A very important thing to remember with regard to the sacral segments is that they supply the back of the limb and buttocks. Why one lays particular stress on that is, that if one is testing sensibility on a patient lying in bed, one is apt just to expose the legs and test down their front aspect for alterations of sensation. By so doing it would be easy to miss a lesion of the cauda equina which had produced sensory disturbance on the posterior aspect of the buttocks or legs. So it is most important, in any examination of the nervous system, to turn your patient over on to his face and examine the back most carefully for alterations of sensation. I want now to indicate what sensory impulses pass by the second neuron, after the first one has ended in the spinal cord. If we represent the spinal cord in a vertical position, we have the fibres which carry epicritic, protopathic and deep sensibility, in different groups, running in together in the posterior roots. The fibres which carry deep sensibility run up in the posterior column on the same side of the cord as that on which they enter, up to the medulla. There is a cell relay there where impulses cross over to the opposite side (sensory decussation) and run up to the optic thalamus in the mesial fillet. With regard to cutaneous sensation there is a re-grouping, thermal sensations go up together, and they cross at once to the opposite side of the cord. All the impulses of heat go together, and similarly all the cold impulses. There is no longer any division into protopathic and epicritic thermal sensations. The painful impulses cross to the other side of the cord as well, immediately on entry. They run up distinct from the fibres carrying thermal sensations, but in close relationship with them. With regard to touch, touch has evidently two alternative routes, possibly more. Impulses of this character ascend partly on the same and partly on opposite sides of the cord. Since that is what happens in the spinal cord it is clear that from an intra-spinal lesion, say involving one half of the spinal cord, you will have a very definite picture, and that picture of sensory disturbance and motor paralysis is known as Brown-Séquard's paralysis. The result of that would be this. Below the site of the lesion, on the same side as it, there will be motor palsy of the spastic type. There will also be on this side loss of deep sensibility, loss of sense of position, and of movement, because the fibres which carry deep sensibility go up on the same side of the cord as that on which they enter, whereas on the other side, that is to say on the side which is not paralysed, there will be loss of perception of heat

and cold and pain, and to a certain extent touch, but not touch entirely. Another variation in sensory disturbance which is produced by a lesion inside the spinal cord is the state of affairs which obtains in syringomyelia. In syringomyelia, as a rule, you find that though tactile sensation remains normal, thermal sensations and painful sensations are grossly interfered with. That, of course, is possible because all these impulses—heat, cold, pain and touch—run along different lines, and why touch is so little affected as compared with heat, pain and cold sensation is because there is more than one route for tactile impressions to pass by, partly by posterior column of one side, and partly by antero-lateral column of the opposite side. In syringomyelia we find there is the formation of a cavity, with overgrowth of the neuroglia round the central canal. That interferes with the fibres carrying heat and cold and pain sensations as they pass across to the opposite side of the cord, and therefore there is produced a characteristic sensory picture in that particular disease. But it is not only in syringomyelia that you have these dissociated forms of sensibility, because, as we have seen, you can get dissociated sensibility with the Brown-Séquard phenomenon produced by a tumour or by hæmorrhage, or by anything of that sort. So because you find dissociated loss of sensation, you must not necessarily conclude that there is syringomyelia, as there are other pathological conditions which may give rise to it.

We must next pass to the optic thalamus. The optic thalami lie on either side of the third ventricle, mesial to the internal capsule, in the floor of the lateral ventricle. You will remember we said that the motor fibres occupied the genu and anterior two thirds of the posterior limb of the capsule, and the sensory fibres lie just behind that, and pass into the lateral part of the optic thalamus. A lesion in the optic thalamus will produce a very definite group of symptoms, which it is important to recognise. The characteristic thing about a thalamic lesion is this: that there is hemiplegia, which, as far as motor paralysis goes, is comparatively transient; it does not amount to anything very much, and rapidly improves. But on the sensory side you find very marked and peculiar phenomena, and they are briefly as follows: First of all, the development of spontaneous pains. These patients often complain of very severe pain along the affected side. That is a very striking feature. Another feature is the loss of sensation on the corresponding half of the body to that on which the pain has developed. This loss of sensation may be very extreme, or it may be comparatively slight. The form of sensation which is most usually affected is deep sensibility. So there develops in these patients a very marked ataxy of arm or leg on the side of the sensory disturbance. In addition to the ataxy, a certain number of them exhibit a very peculiar phenomenon, that is to say, over-action to stimuli which you might describe as being of an affective character. By an affective kind of stimulus, one means a sensation of pain or pleasure, or something of that sort, something which appeals to the emotions, and to that there is an exaggerated response. If you test the hand of such a patient, if it is not very anæsthetic, with a pin, you will find that the individual will draw the hand away quickly, and exhibit signs of the utmost distress, the pain produced being altogether abnormal, and the response entirely in excess of anything which you would expect to find if it were normal. If the hand is put into warm water the patient will experience an exaggerated feeling of pleasure, and so on.

Now we must pass from the optic thalamus up to the cortex, by the third afferent neuron. You will remember that on the cortex of the brain you have the fissure of Rolando, and behind the fissure of Rolando is the ascending parietal convolution in which sensation has its cortical representation. Knowledge of this is of importance in that it assists in the localisation of tumours, or other cortical lesions, because if a person has Jacksonian fits, and if such a fit starts with a sensory aura—tingling in the thumb for instance—you may conclude that the tumour is situated behind the fissure of Rolando, in a position corresponding fairly closely with the thumb area in front of it. Of course it is exceedingly difficult to map out localisation of sensation in the cortex with anything like the accuracy with which you can map out the motor representation, because if you operate on monkeys and animals of that kind you cannot analyse the loss of sensation which results, but you can analyse the movements produced by stimulation of the cortex. And it is interesting that Cushing, a short time ago, performed what is

in fact a human vivisection experiment bearing on this point. He was operating on a brain tumour, and arranged, with the patient's leave, that when the brain was exposed, the administration of the anæsthetic should be stopped, and that the individual should be allowed to come round. That was done, and the post-Rolandic area of the brain was then stimulated electrically with a very moderate strength of current. Every time the current was applied to this region the patient experienced a sensation, but curiously it was always a sensation of movement, not of touch, or pain, or anything of that kind, although no movement was taking place. It was usually referred to the arm, although various parts of the cortex were stimulated. Cushing then tried using a little stronger stimulation, but that brought the proceedings to an abrupt termination, because the patient had a very violent Jacksonian fit, and it was thought inadvisable to continue. It shows that sensation is certainly represented in this part of the cortex, but we are still in the dark as to its exact representation. But as a result of clinical experience there is this to be said: that cortical lesions affect the discriminative faculties of sensation much more than any other form. Pure cortical lesions will practically never produce analgesia: I have to show you now a series of slides which illustrate sensory disturbances that are of common occurrence in disease.

This slide represents the anæsthesia produced by a capsular lesion. The anæsthesia involves the opposite half of the body, but is more marked towards the peripheral part of the limbs than elsewhere. Presently we shall contrast it with hysterical anæsthesia, which is also a cerebral phenomenon, and we shall see that although the area involved is the same, the intensity of the sensory loss differs. In the hysterical case the sensory loss is absolutely uniform, that is to say sensation is uniformly absent, but in this case, which corresponds to a capsular lesion, there is most marked loss in the peripheral part of the limb. Here is a case of hemianæsthesia which is clearing up. The loss of sensation disappears first on the trunk and on the proximal parts of the limbs, but always remains longest at the peripheral part of the limb. The next slides represent hysterical anæsthesias. If you find in a patient a loss of sensation which is very bizarre in its distribution, one which corresponds to no known segmental or peripheral distribution nor to any cerebral lesion, it is almost certain to be hysterical anæsthesia. You know there is a "stocking and glove" form described. The characteristic thing about these hysterical anæsthesias is the very abrupt change from a place where there is no sensation to one where sensation is perfectly normal. That may occur at the wrist, half-way up the arm, or, taking in the shoulder, there may be a fore-quarter affected. But there is always the same sort of abrupt line of transition from places on the skin where there is no sensation to places where the sensation is normal. Here you see an anklet loss of sensation. Such a case shows a distribution of sensory loss that does not correspond to any organic disease at all, either peripheral nerve or spinal segment.

These next slides represent sensory disturbances in tabes. In tabes dorsalis it is very important to investigate sensation very carefully, because long before the ataxic stage develops there are alterations in cutaneous and deep sensation, which are of the utmost importance in diagnosis. And it is important to remember this because of the necessity of establishing the diagnosis as soon as possible. The first slide represents the areas which, in early tabes, are often hyperæsthetic. In these areas an abnormally large response occurs to various stimuli, and we shall see that these are the areas which later on tend so much to become anæsthetic or analgesic. Particularly you notice that the area along the ulnar side of the forearm is affected very early in tabes. Usually you find there a loss to painful sensation. Ulnar analgesia, say in a case of obstinate vomiting, would be quite sufficient by itself to enable you to diagnose tabes dorsalis with gastric crises, quite apart from Argyll-Robertson pupil or absent knee-jerks, for the latter are often present in such cases. Here you see a representation of a case of tabes where the diagnosis would not be difficult, apart from the sensory disturbance, because there was Argyll-Robertson pupil and loss of ankle-jerk on one side, although the knee-jerk and the ankle-jerk on the other were increased. There was arthropathy of one knee, and an area on the front of the chest was hyperæsthetic. There was no analgesia in that case at all.

The next slide resembles somewhat the diagram we saw of a case of anæsthesia of cerebral type

recovering. On this occasion you notice the sensory loss involves both arms and both legs. It is more intense at the periphery of the limb, and the upper area is not sharply defined as we saw it in the hysterical case, but there is a gradual tailing off from abnormal into normal sensation. You find such anæsthesia in cases of peripheral neuritis where there is sensory loss, as, for instance, in arsenical or alcoholic neuritis, whereas in lead palsy there is less likelihood of there being marked sensory disturbances.

I have now a couple of patients to show you. The first is a man, æt. 42 years, who exhibits sensory changes resulting from a lesion of certain posterior roots in the lumbar plexus. His history is that about three months ago, when stooping down, he had a sudden attack of pain in the left buttock. This pain was so severe as to require morphia, and the man had to stay in bed for two or three days at that time. On getting up, he found the leg weak and numb. We are unconcerned for the moment with the motor power in the leg, which exhibits paresis of a lower neuron type. What I want to demonstrate to you is the sensory loss which is present. We will first test his sensation with cotton-wool, and you will notice the tactile cutaneous sensation is only slightly affected in the area supplied by the fourth and fifth lumbar roots of the left side. When we test him with a pin, however, we find he has definite analgesia in that area, whilst his appreciation of pain in the areas of the sacral root supply is perfectly good. Testing him with hot and cold water in test-tubes, we find he has thermal anæsthesia in the same area in which we have noted his analgesia, *i. e.* the skin supplied by the fourth and fifth lumbar roots. This loss of sensation is, however, only relative. Six weeks ago it was almost absolute, so you see his condition is improving. This fact, together with the acute onset, makes a vascular lesion of the meninges almost certain. You may ask why it is that his painful and thermal sensations are more affected than his cutaneous tactile sensation. The reason for this is to be found in the fact that there is a considerable overlap of cutaneous tactile fibres in the posterior roots, whereas those fibres which carry impulses of pain and heat and cold do not overlap to anything like the same extent. The second patient illustrates many of the sensory defects which are found in tabes dorsalis. In this disease the fibres which carry deep sensibility are affected first and suffer most. This patient illustrates that, because, as you see, his gait is extremely ataxic. He walks on a wide base, there is great over-action of the muscles in walking, and he keeps his eyes on the floor. He has educated himself to a certain extent to get his legs into their proper position whilst walking by means of sight. When he is deprived of this aid, walking becomes impossible. He can stand fairly well with his feet together and eyes open, but there is very great unsteadiness when his eyes are closed (Rombergism).

It is a great mistake to think that you cannot do tabetics much good. I have seen them so ataxic that they could not walk at all, yet after patient treatment by appropriate exercises for four or five months, the same patient has been able to walk, even without a stick. It is loss of the sense of position and of passive movements that make this patient so ataxic, but loss of pressure pain sense, another form of deep sensibility, occurs very early in tabetics.

Here is an instrument, known as an algometer, by means of which one may measure the pressure necessary to produce pain. The instrument has a spring with a scale attached, and in a normal person, as you see, pain is produced at a pressure of 5 on the scale, but in this tabetic I can push the spring up to 15 on his leg without evoking any complaint of pain, and I have only to stop then for fear of injuring his skin. This patient suffers also from loss of cutaneous sensation as he has well-marked analgesia in various areas, but the most pronounced change in his case, and in the majority of tabetics in the early stages, is the loss which I have demonstrated in deep sensibility.

January 6th, 1913.

Treatment of Furunculosis.—Schüle advises cauterising the centre of an incipient boil as the best measure for aborting it. He applies a little local anæsthesia before the actual cautery. The first boil is generally too far advanced before it is seen, but the following one treated by prompt cauterisation in its early stage is aborted and the crop is thus destroyed.—*Journ. A.M.A.*, vol. lix, No. 24.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from p. 416, vol. xl.*)

(125)

Dangers of Short-circuiting in the Feeding of Infants.

In my private practice I meet with wasting infants, who immediately after birth have been submitted to the tender mercies of a wet nurse in full lactation. If a healthy infant cannot be expected to short-circuit the colostrum period, how much more improbable is it that a delicate and degenerate infant, on whose life, perhaps, much depends, will be able to dispense with simple lessons in digestion, and learn to run before it can walk.

("Common Errors in Infant Feeding," by Eric Pritchard, M.A., M.D.Oxon., M.R.C.P.Lond., 'Clin. Journ.,' vol. xxxv, p. 263.)

(126)

Food Injuries.

The food injuries of Czerny, his fat injuries, his sugar injuries and his proteid injuries are the old familiar fat, sugar and proteid dyspepsias of Rotch and Holt, but writ large and in capital letters.

(*Ibid.*)

(127)

Something must be done!

Everybody connected with the patient is upset, as indeed they may well be. The first impulse of the friends, equally naturally, is to demand that something should be done, the first impulse of the doctor also is to do "something." Whether what is done is beneficial or not is another question.

("Puerperal Eclampsia," by G. E. Herman, M.B., F.R.C.P., 'Clin. Journ.,' vol. xxxv, p. 289.)

(128)

Successful v. *Unsuccessful Cases.*

But remember that successful cases have a knack of finding their way into print, whereas the unsuccessful one are easily forgotten.

(*Ibid.*)

(129)

When Science fails we must needs fall back upon Conjecture.

Nothing is impossible in science: all science is merely the revelation of truth; truth is the epitome of knowledge, and every day the revelations of science are advancing our knowledge. But, knowledge failing, there is left only conjecture on which to rely—that conjecture which Celsus described as peculiar to the medical art. Still, in conjecture, as expressive of originality, there is generally some measure of interest—a fact of which there is plenty to remind us in connection with the speculative opinions regarding the functions of the thyroid secretion.

("The Treatment of Diseases of the Eye with Thyroid Extract," by Percy Dunn, F.R.C.S. 'Clin. Journ.,' vol. xxxv, p. 299.)

(130)

Slight Errors of Refraction the Cause of Eye-strain.

It is important to remember that it is not the error of refraction that causes the peripheral irritation, but the unconscious correction of the error by the patient. When the defect is great no attempt is made to correct it, as the ciliary muscle can only correct low degrees of astigmation; hence there is no eye-strain.

It is only a cyclopegic that will reveal this unconscious correction; hence, no eyes can be reported as normal unless carefully examined under atropine or homatropine.

("The Manifestations of Eye-strain," by Ernest Clarke, M.D., B.S., F.R.C.S., 'Clin. Journ.,' vol. xxxv, p. 321.)

(131)

Eye-strain as a Cause of Functional Neuroses.

The physician who is called upon to treat a so-called "functional nerve disorder," and fails to eliminate the element of eye-strain, fails in his duty both to himself and to his patient, for there is no functional trouble that may not be due to eye-strain.

(*Ibid.*)

(132)

Soap and Water v. *Antiseptics.*

Plenty of soap and water should be used everywhere, and the house generally kept spotlessly clean. There is no need to sprinkle the room with any antiseptic substance; ordinary cleansing with soap and water is more efficient, and the use of antiseptics may lead to a false sense of security and to the neglect of common cleanliness.

("Practical Hints for Consumptives," by Marcus Paterson, M.B., B.S, and Frank C. Shrubsall, M.D., M.R.C.P., 'Clin. Journ.,' vol. xxxv, p. 332.)

(133)

Effect of Exopthalmic Goitre on Character.

You will see by his description that they (patients with exophthalmic goitre) are persons of hypertrophied conscience, people who are extremely anxious to deny themselves in order to do their duty in every possible way with a sort of restless virtue.

"Exophthalmic Goitre," by W. P. Herringham, M.D., F.R.C.P., 'Clin Journ.,' vol. xxxv, p. 342.)

(134)

Multiple Hard Chancres.

Another thing about extra-genital chancres is that they may be multiple. You should not be put off because there happen to be two or even half-a-dozen or more hard chancres.

("Extra-genital Chancres," by George Pernet, M.D., 'Clin. Journ.,' vol. xxxv, p. 374.)

(135)

Chancres not Necessarily Confined to the Genital Organs.

You must never be put off by position, because primary chancres have been found all over the body in most unusual places. That is no reason why you should not think of syphilis. Medical men, as I have said, often do not think of syphilis at all. A syphilographer always thinks of syphilis, sometimes too much, maybe, but that is a good fault, for it is better to think of syphilis than to overlook it, especially when a chancre is situated about the cephalic region.

(*Ibid*)

(136)

Extreme Youth affords no Protection from Acquired Syphilis.

Children may contract extra-genital sores by being kissed by strangers, or as as a result of being nursed by strange women whilst the mother is ill. A man or a woman who has syphilis may quite inadvertently communicate it to his or her children by kissing them. Therefore you must always be on the look-out for syphilis, not only in adults, but in children too. The fact that the patient is a child must on no account put you off the scent of syphilis.

(*Ibid.*)

(137)

When in Doubt, think of Syphilis.

A good rule to follow is, that where you see things that are unusual and do not fit in with the usual lesions met with in ordinary practice, to think of syphilis, and in many cases something that is unusual does turn out to be syphilis.

(*Ibid.*)

(138)

In Cases of Obstinate Sores, think of Syphilis.

Another point about extra-genital chancres is that a sore place that does not get well under ordinary treatment ought to make you think of syphilis.

(*Ibid.*)

(139)

Truth is not dependent on Authority.

Do not be misled by authority, however respectable or eminent. Authority has done a great deal of harm in medicine.

(*Ibid.*)

(140)

Syphilis, "G.P.I.," and Tabes.

Another curious fact in favour of the syphilitic origin of tabes is, as was shown by Krafft-Ebing, that general paralytics are insusceptible to inoculation with the syphilitic virus. I do not know if the same has been proved in respect to tabes, but probably the result would be the same.

* * * *

All these facts taken together seem to leave little room for doubt that general paralysis and tabes are of syphilitic origin, and that tabes and general paralysis are *per se* as much proof of syphilis as gummatous affections of the skin.

("The Ætiology and Treatment of Tabes," by David Ferrier, M.D., F.R.S., 'Clin. Journ.,' vol. xxv, p. 385.)

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ON FRACTURES.

By T. H. OPENSHAW, M.S., F.R.C.S.,
Surgeon to the London Hospital.

MR. PRESIDENT AND GENTLEMEN,—I am deeply sensible of the honour you have conferred upon me in requesting me to deliver your annual oration, and it is with the utmost diffidence that I have acceded to your suggestion. I feel that narrating is not my strong point: at the same time, when my friend Dr. Armstrong suggested that the subject of fractures would be of interest to the members of your Society, I thought perhaps that my long experience in accident work should enable me to put a few points before you which might be of interest, and when I heard that my friend Dr. Arthur Roberts was your President (? elect) I allowed myself to be tempted, and to assume a position from which by nature I shrink.

Thirty-five years of hospital work in the East End of London (many of them spent at the Poplar Accident Hospital, where I had for many years one of your members which I see before me, Mr. Howse, as a colleague) has suggested to me that I might with advantage recall to your memory some of the changes in diagnosis and alterations in treatment which have taken place.

In my early student days it was no uncommon thing for a surgeon to walk out of the dissecting room straight up into the theatre in order to amputate a crushed leg. He operated clad in a dirty, clot-stiffened garment, which was kept in a cupboard for the purpose, and I have heard a man congratulate himself that the patient lived for six days after amputation of the thigh, and did not die at once from shock or an acute septicæmia.

The advent of antiseptics and aseptics, the life work of Lord Lister, have changed all that, and now it is comparatively rare for any injured leg even to require amputation, and rarer still for any patient to die from acute septicæmia.

But there is another epoch, which also stands out with marked prominence so far as the diagnosis and treatment of fractures is concerned: I refer to the discovery of Röntgen rays.

If you will permit me, I will first bring to your notice a few regions of the body in which our knowledge of fractures has been profoundly modified since the discovery of X rays by Professor Röntgen in 1895, and will then give you very briefly my ideas on some points in the treatment of fractures by operation.

Let us take the elbow first. Who does not remember how exceedingly difficult it is, and always has been, to exactly determine the condition within the joint in a man who has met with some injury to his elbow? The part is swollen, and there is considerable pain on the movements of pronation and supination. One examines the olecranon, and it is apparently intact. There may be some tenderness over the bone, but so there is when the bone has been bruised. An X-ray shows it to be cracked through. In another case we examine the olecranon, and it is detached. An X-ray will show exactly how much displacement there is, and whether any comminution exists. We examine the internal condyle, and it is in place. The head of the radius is intact; it rotates with the shaft of the bone. There is, however, a history of a fall, and there is some tenderness on pressure over the head of the bone. An X-ray shows a split in the head of the bone (Mrs. Chichester), a fracture which no other method of examination could enable us to even guess. We examine the external condyle: it is absolutely normal in shape, but the whole joint on the outer side may be a little tender. We say that it is a contused elbow-joint. An X-ray may show a definite detachment of the external condyle (Bates); it may show a crack of the capitellum, or it may show a tiny little snip of the coronoid process broken off. Each one of these fractures is sufficient to render the joint permanently stiff, and may end in absolute ankylosis. Each is sufficient to set up arthritis, which may limit subsequent movement, and considerably interfere with the patient's future prospects, yet each of these conditions may be mistaken for a severe contusion.

Again in young subjects, given an injury to the elbow, we first ascertain that the radius and ulna are not displaced backwards. If there is a posterior projection of the condyles backwards, and a bulging of the lower end of the humerus forwards, we have been accustomed to diagnose such a case as a separated epiphysis. Careful examination of X-rays shows distinctly that it is exceedingly rare for any epiphysis to be detached, and that in a great majority of cases the condition is not a separation of the epiphysis, but actual fracture on the diaphysial side of the epiphysial line (Wallace Muir). Separation of the epiphyses does occur, and it is essential that if there is separation it should be immediately reduced.

There is one of these fractures to which I should like to draw your particular attention, and it is the fracture of the external condyle of the humerus. In this fracture the external condyle is broken off obliquely into the joint. The fragment may be large or small. It may be displaced upwards, backwards, or forwards. The fracture may be accompanied by fracture of the head of the radius, and a displacement of the head of the radius forwards or backwards may occur. On the other hand, there may be no displacement and no detachment. The bone may unite with simply a little antero-posterior thickening of the external condyle. As regards the movements of the joint, if there is no displacement the movements may be normal. If, however, the bone is displaced upwards at the time of the fracture or subsequently, the forearm will be abducted. If the fragment is displaced forwards, there will be an inequality of the outer border of the humerus, and a depression at its lower end. Flexion of the forearm will here be limited, but the joint can be hyper-extended. An X-ray skiagram, taken stereoscopically, will enable one to see exactly the line that the fracture has taken, and demonstrate exactly the position of the displaced fragment, and will enable one to judge exactly what manipulation is required in order to replace the fragment in its proper situation. If the fragment is fully replaced and is kept replaced, union may result with practically no impairment of movement, but if the fragment is anteriorly displaced, and is not replaced in its normal position, then it is no uncommon thing to find subsequently an elbow-joint where flexion is limited to a right angle, owing to the locking of the prominent displaced external condyle against the anterior surface of the forearm (radius).

The fragment, therefore, should be replaced and placed in a special splint, and another X-ray should

be taken to show that the fragment has been accurately replaced. If it is found impossible to replace the fragment, then open operation should be resorted to, and the bone wired in its proper axis and position.

Some six months ago a young girl, æt. 15 years, was brought to me, who complained that one arm was becoming slowly bent outwards at the elbow, and that she was losing power over her fingers. Examination of the forearm showed that there was slight wasting of the muscles on the inner part of the forearm, and with some wasting of the hypothenar eminence and first interosseous muscles. Examination of the course of the ulnar nerve revealed nothing except that it seemed a little tender near the elbow. On examining the two arms, however, it was distinctly apparent that the left forearm was abducted at the elbow, and made an angle of 155° with the arm. On examining the external condyle there was a distinct tenderness on pressure upon the bone. On carefully grasping the external condyle, it seemed as if the external condyle could be very slightly moved forwards and backwards.

History.—Miss B—, seen June, 1912. Left arm: fractured external condyle, ulnar paresis. Well till 2½ years ago.

Riding bike found the brake would not work so jumped off and fell on arm. Arm bruised and scratched, but attended by doctor and got all right.

Last winter whilst tobogganing in the snow she fell and knocked her arm again. It was considerably bruised but she was again attended by the doctor, who treated it as a contusion and it seemed to get quite alright.

For past six months when practising the violin has complained of aching in the elbow and has seemed not to have the proper use of her fingers. I diagnosed a fracture of the external condyle, ununited, with slight displacement upwards, abduction of the forearm, and pressure by stretching of the ulnar nerve. An X-ray definitely showed an oblique fracture through the external condyle, with little or no displacement and only ¼ in. of separation.

To make a long story short, an accurate diagnosis of this condition would have been absolutely impossible without an X-ray. The complication of paresis of the ulnar nerve, which in my opinion was due to a stretching of the ulnar nerve round the lower end of the humerus, is such a rarity that in all probability it would not have been thought due to a fracture of the external condyle.

Before proceeding to cut down on the fracture and wiring it, which I did on June 26th last, I had made for her a leather splint, jointed at the elbow and embracing the hand, so that pronation and supination are controlled, fitted with elastic traction at the elbow, so as to control any tendency there might be towards eversion of the forearm. (In passing let me say that after wiring this fragment, the tendency to non-union and to subsequent displacement of the fragment is very great, and I have ventured to bring this splint to your notice. It effectually controls the tendency of the forearm to be displaced laterally at the elbow). The bone of this patient is now firmly united, the arm is straight, and the weakness in the ulnar nerve region has disappeared.

This is the second case of paralysis of the ulnar nerve after fracture and separation of the external condyle which I have met with within the past twelve months.

In the second case twenty-five years supervened before the onset of the fracture. Now permit me to divert your attention for a moment to injuries of the wrist.

It is extraordinary how frequently, if an X-ray of a severe sprained wrist is taken, some fracture or dislocation of one of the carpal or other bones is noticed. The most frequently fractured carpal bone to be met with is the scaphoid. It is necessary to be careful not to mistake a dislocation of the scaphoid for a fracture; many observers have fallen into this error. It is rare for the scaphoid to be the only bone broken; the fracture may accompany a partial or complete dislocation of the wrist. Where a dislocation of the wrist co-exists, the symptoms of this fracture are masked. We must always be alive to the possibility of a fracture of the scaphoid. It is always difficult to translate an X-ray of the carpus. The shadow thrown by the scaphoid is rounded at the upper end, and is sometimes separated by a dark line from the lower end. This dark line may easily be mistaken for a line of fracture. A lateral X-ray of the carpus, however, at once disposes of this error. A fracture of the scaphoid should never be diagnosed because a black line crosses it in the X-ray, nor when the portions of the bone appear to be impacted, nor where the

borders appear jagged, or appear to project abnormally. Neither the shape of the bone, as seen in the X-ray, nor changes in length or size are reliable. The only reliable sign is distinct separation of the fragments. In spite of errors of diagnosis, however, the fracture is one of extreme frequency. It is said to occur in 2 per cent. of all fractures. A fracture of the scaphoid alone is not difficult to diagnose; the symptoms are moderately clear. Following a fall on the hand, especially when running, we have pain, swelling, loss of power and limitation of movement of extension, and of radial abduction of the hand. Sometimes an ecchymosis appears, occupying the base of the thenar eminence. The base of the first metacarpal bone will appear to have been displaced upwards towards the lower end of the radius. The most characteristic point of this fracture, however, consists in a filling up of the anatomical snuff-box by something firm. At this spot crepitation may be occasionally elicited. In an absolutely typical case the intra-styloid groove of the wrist is undisturbed, which is not the case where the radius is simultaneously broken.

These X-ray photographs show fractures of this bone. The bone may be notched and cracked, or entirely divided by a jagged line about the middle. The lower portion may be rotated or displaced.

It is possible to differentiate the following facts: Where the fracture is due to direct violence, the fragments may be extremely variable; where the fracture is due to indirect violence, the usual line of fracture is at the junction of the articular portion of the bone with the non-articular, the line of fracture being irregularly transverse.

The prognosis is much worse than is the case with a fractured radius. Occasionally very little disability results. In most cases, however, limitation of movement and pain persist for a long time. Often after apparent recovery so-called rheumatic pain supervenes.

I need not insist how important it is to eliminate the existence of fracture in any sprained wrist, seeing that the ultimate end of a sprained wrist may be considerably aggravated by the presence of a fracture of one of the carpal bones. The lesion itself is insignificant, but the pain and incapacity resulting therefrom are often very considerable. Many of the cases have been found in men who, after an injury to the wrist, have declared themselves incapable of doing further work, and have been classified as malingerers.

Treatment.—In a recent case an attempt should be made to replace the lower fragment, and where this has been done both fragments will rest against the os magnum, and union results. In a long-standing case, however, where pain and loss of power are considerable, a partial resection of the scaphoid may be performed with excellent results, but there will usually persist some slight limitation of movement. This operation however, has not been performed sufficiently often to enable one to make a true estimate of its results. The importance of the scaphoid in the production of Colles's fracture is one which has been considerably overlooked. The scaphoid bone is in direct continuity with the radius. Any force passing down the radius passes through the scaphoid to reach the ground. When, however, in falls on the hand the wrist is hyper-extended, the scaphoid tends to lie on the dorsum of the upper surface of the os magnum, and to rest against the back part of the under surface of the lower end of the radius. With the hand in this position, a slight extra wrench will produce a sprain. If the force is more violent, it is transmitted to the posterior edge of the radius through the scaphoid, and if the angle made by the radius with the hand is 45°, a fracture of the posterior border of the radius is apt to result; and if the force is greater still, the fracture of the radius is complete. If, on the contrary, the impact is received on the thenar eminence, the force is transmitted to the radius through the scaphoid. To produce this, the hand must be inclined to the radial side. Another factor, however, is essential for the manufacture of a fracture of the scaphoid bone; the inherent resistance of the radius must be greater than that of the scaphoid; in other words, the scaphoid must be fixed by the position of the hand, and the force must be transmitted directly through it to the radius, and the radius must be stronger than the scaphoid. When these circumstances are all present fracture of the scaphoid will result.

This fracture has peculiar interest for me because some eighteen months ago, a gentleman, over 50, somewhat osteo-arthritic, with creaking knees and a full habit, consulted me with a history that for the past year he had had severe aching in the right wrist, and about three months previously he had sprained it, and since then the aching pain had

been worse. Examination of the wrist revealed nothing except some limitation of movement and some slight thickening just below the lower end of the radius. I came to the conclusion that in all probability he had suffered a severe sprain of the wrist-joint, but that as he had tried massage and liniment, I thought perhaps if he wore a splint at night and kept the joint at rest, he might be materially benefited. This letter is the next I heard of him.

"DEAR SIR,—I have received your statement of account, being, I take it, for fees in connection with my consultations with you in the early part of 1911.

"You will recollect that the first time I saw you you told me that there was nothing radically wrong with my wrist, and were inclined to believe that the pain I suffered was of a gouty nature. According to your advice, I had a plaster cast taken of my arm up to the elbow, from which a casing was made from the middle of my fingers to the elbow, for which I paid £2 2s. 0d.

"You instructed me to wear this casing for at least six months in order to stop any movement of my wrist, and at the end of that time you practically guaranteed that the wrist would be well.

"This casing was worn for some six weeks, when I found my arm to be getting very thin, and my friends strongly advised me to obtain further advice, which I did, with the result that your diagnosis was found to be absolutely incorrect. I went to the expense of obtaining an X-ray photograph, which clearly showed that the scaphoid was broken in half, and that there was a gap of at least one sixteenth of an inch between the broken parts.

"I understood from the physician whom I consulted that I could either have the bone extracted, or follow his treatment, which was that the wrist should be exercised as much as possible, which is directly opposite to your advice. I was told that if I had followed your advice for six months my arm would have been permanently withered, with no hope of cure in the future.

"The fractured scaphoid, I understand, can never be healed, but I think the better of the two evils is that I have the use of my wrist with some little pain than having an arm permanently withered.

"I have therefore taken no notice of your accounts as they have been rendered, and I think that under the circumstances I ought to be refunded the fees I first paid you, and also the expense I went to in having the casing made as above mentioned.

"Yours truly,"

"*May 7th*, 1912."

I venture to narrate this case as showing how important it is in cases of what appear to have been merely a sprain of the wrist to have an X-ray taken if it is at all possible. My experience would lead me to say that in no instance where the friends of the patient suggest an X-ray is it safe at the present time for a medical men, even if absolutely certain of his diagnosis, to consult the patient's pocket and say it is not necessary. Rather say: "If you wish it, by all means."

An X-ray photograph permits a positive and early diagnosis of the presence of fracture, its exact nature and variety, and number and position of fragments, amount and direction of displacement. In the neighbourhood of joints the condition of epiphyses and the presence of fracture-dislocation are at once determined by a good X-ray.

There are one or two fractures in the leg which I should like to bring to your notice. Let me first deal with the region of the os calcis. What I have said about the advantage of X rays in respect to the elbow-joint applies with equal force to the region of the ankle.

In the old days one recognised that by violent contraction of the tendo Achillis it was possible to detach the epiphysis at the back of the os calcis. One recognised also that, as the result of a fall on the heel, one did get detachment of a large portion of the back part of the os calcis, and that the fracture seemed to run obliquely downwards and forwards. One also has met with cases where the front part of the os calcis seemed to be cracked through, usually as the result of direct violence, and one knew that as a result of a fall from a height on to the foot the os calcis was broken, with consequent extravagant swelling, thickening, filling-up of hollows, pain, and subsequent ankylosis of the ankle and impaired mobility and stability.

In consequence of X-ray examination of many injuries about the heel, one is surprised how much more often the os calcis is damaged than we used to suspect.

The os calcis may be broken by a detachment of the insertion of the tendo Achillis, or it may be

broken by a fall on the heel, the fracture running obliquely forwards and downwards from a point in front of the posterior tubercle. The anterior tubercle may be broken off. It is, however, more particularly to fractures of the body of the bone that I would direct your attention. My attention has been called to this type of fracture by the frequency with which, after the case has apparently recovered, the patient is tormented by an acute pain in the neighbourhood of the external malleolus. This pain very closely simulates the pain which is experienced in very severe flat foot. It is due to continuous pressure upon the external malleolus. This pressure persists after a fracture of the os calcis, and is due to a lateral expansion of the os calcis secondary to the compression of the bone, coincident with the fracture.

Of all fractures of the os calcis, fractures of its body are the most numerous; they represent two thirds of the cases. They are always consecutive to a jump from a height on to the sole of the foot and the degree of the injury varies.

The fractures may be divided into three stages. In the first stage, the weight of the body acting through the astragalus is sufficient to drive that portion of the os calcis which articulates with the back part of the astragalus downwards into the body of the bone. This articular surface, instead of being oblique, becomes horizontal. The patient has fallen on one foot or both, and may or may not have perceived a cracking. It may be possible for him to walk, but with some *pain*. Some hours afterwards there is *swelling* and *ecchymosis*. There is discoloration and swelling below the external malleolus. There is no other deformity, and no luxation of the foot. The fibula is intact. There is no abnormality of the foot. The ankle-joint movements are normal. There is no pain on pressure over the outer part of the tarsus. No abnormality is seen at the back of the heel. The arch of the foot is normal. The only place where there is any swelling is below and behind the external malleolus. Here the swelling may be very tender on pressure, and quite hard and firm, and may to a great extent occlude the outline of the external malleolus. An X-ray shows a descent of the posterior superior articular facet, and a notched line of fracture somewhat semicircular in outline within the body of the bone. The vertical diameter of the bone is decreased.

In the second stage, the depression of the articular surface continues and the bone is expanded on the inner side, falling upon a hollow below and behind the internal malleolus. In this stage, owing to the swelling on each side of it, the heel uppears to be shortened. The normal arch of the foot is thus effaced, and the case resembles one of traumatic flat foot. The descent of the astragalus in these cases is considerable, and it rotates so that the head is actually higher than the body. This rotation of the astragalus stretches open the astragalo-scaphoid joint. The trabeculæ of the os calcis are split up by fractures which radiate in all directions. The upper surface of the os calcis, instead of presenting an eminence, presents a concavity. The axis of the os calcis is not altered, its anterior end is not displaced.

The third stage of this fracture may be described as a total crushing of the bone. The profile of the foot is entirely altered, the heel is thickened and rounded, bony masses expand upon each side of the malleoli, and the foot presents extravagant flat foot; its under surface may even be convexed. The transverse diameter of the os calcis is enlarged, and the fracture is quite apparent. The front part of the astragalus projects on the dorsum of the foot.

It is to the subsequent treatment of the deformity of this fracture that I more particularly wish to draw your attention. The thickening of the bone, pressing continually as it does on the inner surface of the external malleolus, causes a grinding pain especially when walking. So severe may this pain be that it may entirely prevent a patient taking any exercise or doing any work.

I have had many—about a dozen—cases of this nature under my care during the last three or four years, and the similarity of this pain to that experienced in severe flat foot has led me to conclude that removal of that portion of the outer surface of the os calcis which presses on the external malleolus by operation would be beneficial. I have operated upon about a dozen cases of this nature, and in almost all the result has been perfect. In the rest the pain has been considerably relieved, in not one has a complete failure resulted. One gentleman who was an officer in command of a marching regiment in India, and who in consequence of being unable to march was within a measureable distance of being called upon to resign, writes that he is

able to march all day long without the slightest fatigue or pain. This operation I can commend to the members of your Society with every confidence.

The operation consists in cutting down on the outer surface of the os calcis, and chiselling away freely sufficient bone to avoid this pressure against the external malleolus.

Let me now refer for a few moments to the ankle-joint. I remember in the old days that one had five injuries of the ankle-joint: a fracture of the fibula, a fracture of the internal malleolus, a Pott's fracture (which was a combination of the two or with a rupture of the internal lateral ligament), a Dupuytren's fracture, where the inter-osseous ligament was supposed to be ruptured, and the astragalus to be shot up between the lower end of the tibia and fibula, and a sprained ankle.

I do not remember to have ever seen a case of genuine Dupuytren's fracture as we knew it in the old days—that is, where the astragalus has been completely displaced upwards, and lies between the unbroken lower ends of the tibia and fibula. A partial rupture of the ligament is not uncommon, but it is always associated with a fracture of the fibula, either low down or high up.

The clinical point in connection with these fractures, however, which I would like to bring to your notice is more particularly to deal with the displacement of the foot backwards, or of the tibia forwards. Repeated examination of X rays of such fractures where the foot is displaced backwards has disclosed the fact that there exists a vertical fracture of the tibia.

Although often a simple complication of a fracture of the malleolus, this particular fracture of the tibia, where a vertical portion of the posterior surface of the lower end of the bone is detached, is so frequently seen that it may well be considered a definite pathological entity, because of its frequency, because of the deformity, and because of the gravity from a prognosis point of view.

If one refers for a moment to the anatomy of the foot, one sees that the under surface of the tibia is concave, and that the posterior part of it projects well behind the astragalus. The function of this portion of the under surface of the tibia is to prevent displacement of the astragalus backwards, and it is readily understood how easily, when this portion of the tibia is broken off, the foot can be displaced in a posterior direction.

This fracture is usually complicated with the fracture of the external malleolus, and is the more difficult to diagnose in consequence. Its frequency is great; more than a hundred cases are published. The gravity does not appear to depend upon the size of the fragment. Backward displacement of the foot may be perceived where the fragment is small, as well as in the case of a large fragment, which may be so large as to have torn off a portion of the internal malleolus. The fragments are of every size, from a tiny microscopic piece of the internal malleolus up to a large oblique fragment. As in X rays, the external malleolus may also be broken, either at the base or obliqely. The internal malleolus is often broken at its base, but it may remain intact. It is frequently cleft vertically, and causes antero-posterior enlargement of the internal malleolus. It is curious that this displacement may take place either when the fall has been on the flat foot or with the foot extended.

The symptoms of this fracture are somewhat difficult to verify, because of the concomitant fracture of the external malleolus, but they should always be looked for.

It will be seen on careful examination that the lower end of the tibia is a little displaced ourwards and forwards on the tarsus. This deformity is typical, and the difficulty in reducing it and in maintaining the reduction should lead one to suspect the fracture; so that, if with the presence of a malleolar fracture, the reduction of the foot is difficult and is not easily complete, the presence of this vertical fracture must be suspected. By grasping the heel and pressing firmly on the front of the foot, and making steady downward traction, the fibula will be slowly or suddenly drawn forward. If the foot be now dorsally flexed, the projection of the lower end of the tibia will disappear. When this reduction has been effected, the foot should be maintained in a right angle, with the leg on a dorsal splint with a foot piece.

If the tibia has been completely replaced, there should be no depression beneath the lower edge of its anterior surface. The tibia should make a direct flat surface with the front of the astragalus. If there is the least projection of the anterior edge of the tibia it must be removed by very forcible dorsal flexion, and the foot must be put up at an acute angle. Any projection of the lower end of the tibia which resists reduction is very strong pre-

sumption of the presence of this particular fracture. It follows that the presence of this fracture is very difficult to diagnose unless an X-ray has been taken.

The line of treatment should be, if possible, to replace this detached fragment, so that the contour of the lower end of the tibia may be restored as far as possible, in order that the movements of the ankle-joint may be normal, and the astragalus again obtain the posterior support which will prevent posterior dislocation.

If this fracture is not replaced, we have arthritis and partial dislocation as a result. Not only does this detached fragment fail to support the astragalus, but it forms a projection of the posterior surface of the tibia, which interferes with the action of the flexor muscles, and so limits plantar flexion of the foot and toes, whilst the anterior projection of the tibia also interferes with the action of the extensors in front.

The prognosis of these fractures, if not efficiently treated, is grave.

A radiograph, if obtainable, at once solves our difficulty, but a radiograph is not always to be obtained. In the absence of a radiograph, therefore, it is almost wiser to presume its existence if there is the slightest tendency of the tibia to ride forward, or of the foot to be displaced backwards; under these circumstances the foot must be put up at an acute angle; in other words, the heel must be kept well down.

It is curious that at the London Hospital we have for years carried out this treatment empirically. We treat our fractured fibulæ and Pott's fractures on an outside splint and a foot-piece, and carefully stretch the heel well down, so that the foot is kept at all events at a right angle, and in many cases even at a little less than a right angle.

Since the advent of X rays, one has been able to verify the fact that this posterior fragment of the tibia exists in one in ten of every fracture about the ankle-joint.

The first indication for treatment, therefore, is to reduce the displacement of the foot, and the second indication is to pull down and pull forwards the posterior fragment of the tibia by forcible dorsi-flexion of the foot.

For this, vigorous traction upon the heel is essential, and forcible dorsi-flexion. The reduction of the displacement is not complete as long as there is the least projection of the lower end of the tibia forwards. When this has been completely reduced, the foot must be flexed at an acute angle.

If the fracture has not been replaced, and the bone has been allowed to unite with the lower end of the tibia projecting, then operation must be performed, and the projecting lower edge of the tibia removed.

This very day I have had sent to me a patient who was caught between a runaway motor-bus and some railings. He was taken into a country hospital, where he was detained for a fortnight with a so-called contused ankle. He has enormous antero-posterior thickening of the lower end of the tibia, with projection backwards of the bone underneath the flexor tendons, and a projection forwards of the anterior surface of the tibia. He is unable to flex the toes, has limited dorsi-flexion, but can walk on the foot fairly well, but there is this limitation of movement of the ankle.

Now permit me for a few moments to deal with the subject very briefly of the operative treatment of fractures. In the first place, there are certain fractures where it is extremely unlikely that any operative procedure will be requisite: for instance, such fractures as greenstick, fractures of the ribs, fracture of one bone only of the leg and forearm where the adjacent bone acts as a splint, and fractured clavicle usually.

On the other hand, there are certain fractures where operation is imperative; for instance, where the fracture is accompanied by division of a nerve or vessels an operation may be necessary in order to unite the nerve, or for the ligature of the vessel. Here, if it is likely that there may be difficulty in keeping the bones in good position, wiring or plating of the bones may be resorted to.

Again, in compound fractures, unless the wound is microscopic, and there is every reason to think that it is uncontaminated, the whole part should be laid open and thoroughly cleansed with carbolic lotion. Any loose fragments should be removed. Any fragment which seems likely to project or to be very mobile should either be removed or wired. If the fractured ends of the bones seem to be easily replaceable in good position and remain there, plating or wiring is not necessary. Often, however, in severe compound fractures, it will be deemed advisable and unattended with but little extra risk to plate the broken ends.

In all other fractures the real indication for open operation is failure to obtain good position and union by palliative means. Experience, however, has shown that palliative treatment is of varying value with different types of fractures. Let us divide them as follows:

Where open operation from the first is the most likely method of treatment whereby to obtain good union and perfect result as regards function:

In this category we may include (1) fractures of the patella; (2) fractures of the olecranon with separation; (3) some fractures where small fragments are detached and involve joints; and (4) fractured acromial end of clavicle.

The patella.—Fractures of the patella are of two kinds. One is caused by direct violence, which produces a stellate fracture. This fracture unites firmly without any difficulty. The ordinary fracture of the patella, however, is the transverse one, which is produced by muscular violence or operation, and there is always more or less separation of the fragments. Provided the patient can stand any operation at all, this fracture should always be wired. There is often tilting of the fragments, and inclusion of portions of aponeurosis between the divided ends of the bone. These two conditions prevent the bone fragments uniting firmly by bone, unless they are brought accurately in apposition and wired together. Treatment by strapping, by Malgingne's hooks, or by wiring circumferentially, as Mr. Barker did it, is out of date. For many years I wired the patella by means of a long peroneal needle inserted through the quadriceps patella and brought out through the ligamentum patellæ. A double silver wire was withdrawn with the needle, and the two fragments of the patella tightly squeezed together when the wire was tightened. Bony union resulted. X rays have shown me, however, that in certain cases tilting of the upper fragment is not overcome by this method. It is far better, therefore, to make a vertical incision, drill the bones, and wire them together. The wire should not be passed completely through the posterior surface of the bone. It is well to use two wires side by side in case one gets detached.

Fractures of the olecranon.—If there is no separation, a flexible splint may be applied to the anterior surface of the arm and forearm, the elbow being slightly bent, massage being applied at once and continuously. If, however, there is any separation, an incision down to the bone and one wire is sufficient to approximate the fragments and to effect complete union. Immediate operation should be done, because the fragments are often tilted, widely separated, and portions of aponeurosis are often interposed between the fractured ends.

Open operation upon the olecranon is a simple and safe procedure. It permits almost absolute restoration of the joint surfaces, which prevents ankylosis, and it permits early passive movement, which facilitates early restoration of joint function.

In fractures about joints, if a stereoscopic X-ray shows a detachment of a portion of the bone, which is likely to lie loose or to be abnormally placed within the joint, in my opinion the joint ought to be opened and that portion of the bone removed.

Another category where operation may be required, but is less likely to be called for, includes (1) fracture-dislocations; (2) separation of processes where muscles are attached; and (3) T-shaped fractures into joints.

The whole subject of fracture-dislocation is one of great difficulty. It is in the shoulder that these fractures most often occur. It is obvious that the dislocation must be reduced, and the fracture if possible re-adjusted. The arm must be put up in that position which enables the fragments to be retained in apposition. It may be either fully abducted from the chest, partially abducted, strapped to the side with or without a pad in the armpit, or put up on a U-shaped splint.

Granted that the dislocation has been reduced, and that there is no extravagant deformity of the upper part of the bone, palliative treatment may be confidently relied upon. It is obvious that should the detached head of the bone not be replaced, and should there be evidence of pressure by it upon some nerve, viz. the musculo-spinal nerve, it ought to be removed (X-ray vergette), but even after removal of the head of the bone, pain and limitation of movement will persist, although theoretically perfect movement ought quickly to be restored. Absolute recovery of perfect union of the shoulder-joint does not, in my opinion, entirely depend upon the accuracy of the reposition of the detached fragments. I believe there is often reflex spasm or paresis secondary to the injury, and that the limitation of movement is due to this cause, and not to the fact that the re-united head

is not exactly of normal shape; in other, words I consider that a certain amount of limitation of movement is almost inevitable after a severe fracture of the upper end of the humerus, even though the fragments are removed or cut down on and pegged, and the normal shape of the bone thereby reconstituted.

Pegging, wiring or plating the upper end of the humerus is a serious surgical procedure not lightly to be undertaken. The unfavourable results are not published—you cannot expect them to be published. It is impossible to ascertain the exact results as regards movement, or how often it is necessary to remove the plates and pegs because of their becoming loose owing to rarefaction of the bone. For my own part the rotation of the head must be very severe to induce me to open the joint and plate the bone or remove the head.

With regard to separation of processes where muscles are attached—speaking of the shoulder—the separation of the upper part of the great tuberosity is the common detachment. Speaking for myself, I should be inclined to have the arm put up in a fully abducted position and kept in that position in plaster-of-Paris; but should an X-ray clearly indicate that there is separation of the upper part of the great tuberosity only, there is no reason why an incision should not be made down on to the upper end of the humerus, and the detached portion of the great tuberosity fastened in position by means of a screw or ivory peg.

Detachment of the tubercle of the tibia also is a fracture which may present considerable separation, and some difficulty may be experienced in bringing the fragment into apposition with the portion of the bone from which it has been torn. Here, also, the fragment may with advantage be pegged down.

The subject of T-shaped fractures into joints is also one of great difficulty. Each case must be treated on its merits. An X-ray must be taken in several planes and stereoscopically. The X-ray must be clear and good; an imperfect X-ray is worse than useless. The exact position of the fragments must be determined, and the amount of disability experienced by the patient must be taken into consideration.

This X-ray shows you a case where, after a split of the tibia, the patient was wholly unable to stand and walk owing to separation of the articular surfaces of the tibia and displacement of the outer tuberosity. My colleague, Mr. Furnivall, asked me to see the case, and I advised that he should split the patella transversely, take out a wedge from the centre of the upper end of the tibia, and wire the tuberosities of the tibia together. This he did with an excellent result, and the X-ray shows that whereas before the operation the two articular surfaces of the tibia projected beyond the edges of the corresponding condyles of the femur, the articular surfaces are now in accurate apposition. The patient has free movement and perfect power in the leg. This X-ray shows a detachment of a portion of the external portion of the tibia. It illustrates how the outer edge of the external condyle of the femur acts as a wedge in the splitting of an oblique portion of the tibia.

The above-mentioned fractures only constitute exceptional fractures. There remains the immense class of transverse and oblique fractures of the shafts and other parts of the long bones, where, as a rule, palliative measures will, if they are given a fair trial, succeed in replacing the bones in good position and in causing firm union to take place.

I maintain that by means of accurately fitted properly padded splints good apposition of the greater majority of fractures may be maintained, and that union will result if the part is kept at rest. I maintain that splints are necessary in order to keep a fractured leg or arm in good position. It may be easy to put the bones in good position, but splints are necessary in order to maintain them there. Past masters in the art of wiring non-united bones say the art of setting a fracture with splints is a myth. My experience would go to prove that the position of the broken bones is improved after twenty-four hours' rest on a properly adjusted splint, and that the improvement is hastened and increased if a certain amount of traction is applied to the foot. Certain French surgeons say they never use splints. My experience is that abroad none but the good results are ever shown to strangers; even the surgeon himself is not allowed to see his own failures. The teaching of Lucas Champonniere is unreliable in my opinion. It certainly does not accord with my experience. Extension of the foot cannot be maintained unless the foot is strapped to the foot-piece of a back splint. With the foot thus fixed, a continuous

steady traction can be made upon the leg, the muscles relax, and any overlapping of the fragments is slowly remedied, so that at the end of twenty-four hours the position of the fragments will often be perfect, although the shortening may not have been quite reduced at the time that the leg was put up. The pen-nib fracture of the tibia, which often gives trouble, can easily be controlled by the pressure of a pad on the front of the leg above the point of fracture when the foot and the knee are fixed. Treatment of fractures by massage and without splints is followed by non-union, and plating or wiring of the tibia becomes necessary. The rage for massage also necessitates a continuous intermission in the application of rest. Massage is applied, and the splints are removed in order that it may be applied. It is exceedingly difficult to get massage done without having the fractured limb moved. Massage is translated to mean passive motion and massage. In the early days of a fracture there should only be the gentlest massage, without movement of the fractured bones, and there should be no passive movement of the joint. Massage does wonders for the limb; it has revolutionised the after-results of fractures, but only gentlest massage and no movement should be allowed in the early stages of a fracture. In my opinion it is the want of confidence in the effect produced by properly adjusted splints which is responsible for the great increase in ununited fractures at the present day, and it is the belief in the necessity for vigorous daily massage which leads to distortion and non-union, with subsequent necessity for operative interference.

It is true that, given carpentering skill on the part of the surgeon and an accurate knowledge of anatomy, with the patient fully anæsthetised, every part wrapped up except the part actually to be operated on, every assistant and every nurse properly gowned, gloved, masked, and covered as regards the head, it is possible for bones to be plated with impunity, which under other circumstances may be followed by fatal septic osteomyelitis. To make sure of this result it is absolutely essential that no single finger of either the operator or any assistant should be put into the wound, instruments being used for everything. The instruments should be kept in either 1 in 60 carbolic, or dipped in boiling water every few seconds before operation.

It does not necessarily follow, however, because it is possible for a man who is always operating to put twenty-seven screws in a man's leg with impunity, that it is therefore possible for every man to do it, or that it is advisable that it should be done in any but very exceptional cases and never without the most careful deliberation. Not only are screws and plates foreign bodies, but with the most extreme precautions it is exceedingly difficult to avoid their contamination. Frequently they have to be removed, because of failure of the soft parts to close over them, because they give rise to irritation, because they become loose, or because of sepsis.

Here is an X-ray which shows that a steel plate may actually be broken across and non-union of the fracture result, because instead of relying upon a proper splint appliance or plaster case wherewith to keep the fractured ends at rest, reliance has been placed upon the steel plate, and the patient has been allowed to move about too early.

Here is an X-ray of a fractured tibia and fibula which was broken in September, 1910, was wired, and was wired again in February, 1911. The wires ulcerated through, and had to be removed. The leg was put up in plaster and failed to unite. The patient came to me in November, 1911, with the fracture still ununited. In this particular the plaster-of-Paris had not been taken far enough down the leg, with the consequence that every time the toes were pressed on it moved, and a tilting of the fragments at the seat of the fracture resulted. I put up the leg in plaster-of-Paris, including the ankle, and in three months the bone was firmly united, and the patient was walking about on it without the aid of any support.

I cannot too earnestly advocate thorough treatment by rest by means of properly adjusted splints.

I have brought here to show you to-day a boy who on Friday last was admitted into the London Hospital suffering from an oblique fracture of the left thigh-bone. You see him to-day walking about. Here is an X-ray which shows the obliquity of the fracture. He is at this moment walking on his tuber ischii; there is no pressure on the fracture or on the heel. The appliance he is wearing transfers the weight of the body direct from the ground to the tuber ischii, and it is possible to make any ordinary case of fracture, either of the femur, tibia, or ankle, walk within a few days of the date of fracture. Moreover, the exercise of walking is continual massage, floods the part with blood, and facilitates union, at the same time that the weight of the leg acts as an extending agent. This splint enables a patient who has broken his leg to be up walking about within a week.

It is now some few years since I first showed this splint at the Clinical Society. This splint is not only useful for fractures; I have quite half a dozen Charcot's knees, who are walking about, and who have been materially benefited by it.

January 13th, 1913.

LECTURES ON NEUROLOGY.

Delivered to Post-Graduates at St. Bartholomew's Hospital.

By C. M. HINDS HOWELL, M.D., F.R.C.P.,
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LECTURE IV.

(*a*) ELECTRICAL TESTING OF MUSCLES.
(*b*) LUMBAR PUNCTURE.

GENTLEMEN,—It is in diseases of the lower motor neuron that the electrical testing of muscles becomes of considerable importance. The upper motor neuron terminates, as you remember, in connection with the anterior horn-cell, and the lower motor neuron consists of the anterior horn-cell and the peripheral motor nerve-fibre. When this neuron is the seat of an acute lesion, characteristic changes occur in the electrical reactions of the muscle, which constitute what is known as the reaction of degeneration. In chronic disease of this neuron, as, for instance, in progressive muscular atrophy, the muscles gradually lose their excitability, but do not give a typical R.D. as it is called.

It is important first of all to recognise the normal reactions in a healthy muscle. I will show you how the muscles react in a normal individual. You test the reaction of the muscle to faradism and to galvanism. We will first of all test with the faradic current. For this you use an induction coil, and you have a key-electrode, along which the current can only pass when you press the button. So long as the current passes the muscle goes into tetanus. If the current is broken, the contraction stops at once. This is the normal response. If you compare that with the muscular response which takes place to the galvanic current, or the constant current, you will find that it is totally different, because, with the latter, the muscle contracts only at "make or break" of the current and not at all during its flow. The response to this kind of stimulus takes the form of a brisk twitch; it is not a sustained contraction. With a small current, 3·5 milliampères, a contraction takes place beneath the cathode only on "make," but if the strength of the current is increased there will be another twitch at "break." If I reverse the current, I now have the anode over the muscle and I still get a contraction on "make." With the anode the contraction is much smaller than it was with the cathode, although the strength of current remains unaltered. The fact that the cathodal closing contraction is greater than the anodal closing contraction is represented thus: KCC > ACC. The patient I now show you has weakness and wasting of the small hand-muscles; there is also paralysis of the vasomotors, as the hand is congested and cold. The wasting involves the thenar and hypothenar muscles, the interossei and also the extensors and the flexors of the wrist and fingers. We will first test with faradism. You see that although this puts the triceps into contraction, you have no response, or at all events, very slight response, in the extensors of the wrist and fingers. The faradic response is practically lost. The extensor of the third finger is still a little active, but the other extensors do not respond at all, nor do the flexors or intrinsic hand-muscles. Loss of response to the faradic current is one of the characteristic signs of the reaction of degeneration. We will compare the response which we get with galvanism in this case to that which we obtained with the normal patient just now. The extensors are now reacting, but instead of brisk twitching, the contraction in this case is very sluggish. The excitability is also decreased because we have to use 15 milliampères of current to get a response at all. Also we now find that ACC > KCC: that is to say there is complete R.D. with polar changes present in this case. It does not really matter whether polar changes are present or not; the important thing to notice is whether the contraction is brisk or whether it is sluggish to galvanism. But when polar changes are present you have the complete reaction of degeneration. You may ask what is the use of these electrical reactions? They are useful both for diagnosis and also prognosis. In some cases of tabes, for instance, and alcoholic neuritis, it is difficult to say which disease is present: in both cases the knee-jerks are absent, there is alteration of sensation, weakness of the legs, etc., but in a case like that the electrical reactions would be very important, because in the peripheral neuritis you have reaction of degeneration, whereas in the tabetic you would not have that. With regard to prognosis, the electric reactions are of more importance because

you can judge to a certain extent from the electrical reactions as to the severity of the lesion present, say, for instance, in a case of facial palsy. Supposing at the end of a week you have no great alteration in the electrical reactions, although paralysis remains, you can give a fairly good prognosis with regard to rapid recovery, whereas if there is very marked alteration in the electrical reactions at such a date as that, you can be fairly certain it is a severe case with which you are dealing, and that recovery will be slow, if it takes place at all. As time goes on, in a severe case, the muscles refuse to respond to either galvanism or faradism, and when the electrical reactions are completely lost, the point arises whether recovery in such a case can take place or not. It can. It does not follow that because electrical reactions have entirely disappeared that recovery will not take place, but the prospect of it doing so is not particularly good. It does occur, however, on occasion. But it means that you have a very severe case to deal with then, and that the recovery will be a slow one if it occurs at all. So these reactions, which are easy to do, are of considerable importance, both in diagnosis and from the point of view of prognosis. They are also of importance in deciding what form of electrical treatment to order, as you should order as a rule that form—either faradism or galvanism—to which the muscles respond best.

(*b*) *Lumbar Puncture.*

The next thing I want to speak about this afternoon is the question of lumbar puncture. With regard to this procedure, there are one or two points to be borne in mind concerning the anatomy of the region in question. Here I have a spine from which the laminæ have been removed, and with the spinal cord and cauda equina exposed. It will illustrate several of the points on which I wish to lay stress. In choosing your site for lumbar puncture you will, of course, choose a part where there will be less risk of doing damage than elsewhere. So you enter the spinal canal below the termination of the spinal cord. In the adult that level is at the lower part of the first lumbar vertebra, and below this level the spinal canal is filled in with the nerve-roots that you see here, which constitute the cauda equina. In the child the spinal cord extends lower than the first lumbar segment. It is quite safe to enter the needle into the spinal canal between the fourth and fifth lumbar vertebræ. What is the landmark by which you can recognise that point? If you take the upper level of the iliac crests and draw a line joining the two across the back, that line will pass through the tip of the fourth lumbar spine. So you take the nearest spinous process to this transverse line and mark its position under the skin. Then having cleaned up the skin in the ordinary way and boiled your lumbar puncture needle, you are ready to perform the puncture.

What about the position of the patient? There are two positions possible, and certain circumstances determine you in your choice of one or the other. Either you can have the patient sitting on the edge of the bed leaning forward in an upright position, or he can be lying on his side. You do not require a general anæsthetic for this procedure, except in the case of children, and for them it is necessary because they may suddenly make a movement when you have put your needle into the spinal cord, and it may break in. A local anæsthetic may be used—ethyl chloride, for instance—but as a matter of fact it is more unpleasant to have the skin frozen than it is to have the puncture made without it. The upright position is only suitable if you have first of all a patient who is not nervous (and usually patients are more or less nervous), and secondly, if the patient has not any marked sign of increased intra-cranial pressure. If you are dealing with a case of cerebral tumour with severe headache, where you have made up your mind to try to relieve the headache by drawing off cerebro-spinal fluid you must have the patient lying on the side. As a rule you find the lateral position is most convenient. The knees are drawn up somewhat, and the back is slightly arched; moderate flexion will do.

With the patient in the lateral position as described, insert the needle a little to the left or right of the middle line by the side of the spinous process, pointing a little upwards and towards the middle line. You will encounter, after the skin, a firm resistance from the ligamentum subflavum, that forms a dense line of ligaments between the laminæ. The resistance is so great that at first you may think that you are on bone. By moving about the point of the needle you can easily tell whether it is on bone or on the ligament. If on the latter you press on, and the needle goes through

that, and next it goes through the dura mater. You have the stilette *in situ* in the needle, otherwise the bore of the needle is apt to become blocked. After the point of the needle is within the dura, remove the stilette, and have a test-tube ready to take the fluid. You must not exert suction. If cerebro-spinal fluid does not escape you can sometimes start the flow by getting the patient to cough. This raises the pressure of the fluid and may be all that is necessary to start off the flow: The commonest cause of a "dry" puncture is failure to enter the needle within the dura. Are you likely to damage the roots of the cauda equina? You are not, because in 50 per cent. of the cases the roots of the cauda equina lie against the anterior part of the dura, and if you go through gently, feeling each resistance as you go through it, you will not jamb the needle against the body of the vertebra in front. In a child there is a danger that you may do that, and stick the needle into the intervertebral disc in front, or into the body of the vertebra. In any case as a rule the roots are much longer than the canal in which they lie, as a result of which they are not taut, and as the dura bulges in front of the needle it pushes any root which may be lying posteriorly on one side, and when the needle goes through it does no damage. But even if it hits a posterior root you are not likely to produce any ill-effect beyond a momentary stab of pain. If you get pure blood coming from the needle and nothing else you may be sure you have not got within the theca, that is to say within the dura mater, at all, and the blood is almost certainly coming from an extra-dural vein. If that is so you must remove the needle and go in on the other side of the middle line. If the fluid that escapes is tinged with blood this may be due to one of two causes: either a vessel has been injured and is bleeding within the theca, or else the cerebro-spinal fluid is itself blood-stained, as is the case, for example, in cerebral hæmorrhage. In the former case the fluid becomes clearer as it flows, and that first collected must be discarded. In the latter case the tint remains the same throughout and affords valuable information. Next, if the fluid tends to spurt out, which means that it is under considerable pressure, only let it escape gradually. The danger is that if you are dealing with increased intra-cranial pressure, and you draw your fluid off very rapidly, the medulla may be forced down into the foramen magnum. At post-mortem examinations of cases where such increased pressure has been present, you usually see a "pressure cone" formed by compression of the medulla and tonsil of cerebellum into the foramen magnum. If, under these conditions, you draw off fluid, the medulla may be forced down harder than ever, and as a result of that there may be respiratory failure. Many patients with cerebral tumours die suddenly from respiratory failure, as the respiratory centre in the medulla seems particularly susceptible to alterations in pressure. The heart is not affected to anything like the same extent. If you draw off a large amount of fluid very rapidly when the pressure is high there may be respiratory failure in consequence, and for that reason atropine is sometimes given before the puncture. If, however, you only allow the fluid to escape gradually, there is no great probability of any untoward result.

How much fluid should be removed? Usually as much as will come readily; when the flow begins to be definitely affected by the respiratory movements it is well to stop withdrawing it. The patient should remain in bed for some hours at any rate after the fluid has been withdrawn as a precautionary measure. Headache, sometimes severe, is complained of where there has been no increase in intra-cranial pressure, but on the other hand the headache due to increased pressure is often relieved by the operation.

What is the character of the normal fluid? I have some here which was taken off this morning, and you will see it is a perfectly clear fluid in the normal condition. It is of low specific gravity, and alkaline in reaction. It contains a trace of protein identical with serum globulin, and it also contains a trace of sugar. This is detected by pouring off the fluid after boiling with Fehling's solution, when you see the red cuprous oxide deposited at the bottom of the test-tube. In addition it contains a trace of urea, inorganic salts and a very few cells. If you take the normal fluid and examine it under a high-power lens you will find less than one cell to each high-power field. To show you the normal quantity of protein content I show you some cerebro-spinal fluid which has been boiled: you see there is just a slight turbidity compared with the normal unboiled fluid.

Now what are the indications for making a lumbar puncture? It is useful both for diagnostic and for

therapeutic purposes. Let us consider its diagnostic value first of all. First in acute disease associated with "meningism," that is to say where there are symptoms which might be due to meningitis, but where there is a possibility of some other disease being present, and giving rise to the symptoms. Pneumonia, enteric fever or influenza are examples that readily occur to one in this connection. They may be accompanied by cerebral symptoms which suggest meningitis, and lumbar puncture alone will give you definite information as to whether meningitis is present or not. Secondly, there are definite meningitis cases where you want to make a diagnosis as to the nature of the organism which is causing the symptoms, pneumococcus, or tubercle, or meningococcus. Thirdly, it is of great value in the diagnosis of syphilitic or parasyphilitic disease (tabes and general paralysis of the insane) of the central nervous system. Any of these conditions may, in their early stages, give rise to very great difficulty in diagnosis. The difference between incipient general paralysis of the insane and neurasthenia is sometimes very difficult to be certain about from simple clinical examination of the patient. But in such a case examination of the cerebro-spinal fluid is practically conclusive. Certain early cases of tabes, again, give a great deal of difficulty in diagnosis. A person with gastric attacks whose reflexes are normal, and in whom there is very little to make you suspect tabes, may have his condition diagnosed by this means. And in syphilis of the central nervous system, say in a case of paraplegia, which may be due to gumma or to a syphilitic condition of vessels, it is very important for you to make a diagnosis between that and spinal caries, or tumour or something of that kind, and you will obtain very important information as to which is present by examination of the cerebro-spinal fluid. To distinguish between syphilis of the central nervous system and general paralysis of the insane is sometimes very difficult, and although you cannot do it with absolute certainty by examination of the cerebro-spinal fluid, yet you can get in that way information which may aid you very materially in the diagnosis. We will refer presently to the therapeutic value of lumbar puncture.

Having obtained the fluid, what is to be done with it? If you possess a centrifugalising apparatus it is well to centrifugalise it, but in any case first examine the fresh fluid in a Thoma-Zeiss blood-counting apparatus, because by so doing you will be able to form an idea of the number of cells present per c.mm. of the fluid, and that is important in such conditions as tabes and general paralysis. Increase in the cells is pathological, and may mean acute inflammation, tubercle, parasyphilis, or syphilis. Next, make a film, preferably from the centrifugalised fluid, and stain and then examine the cells. Polymorphonuclear leucocytes occur in post-basic meningitis, meningococcal meningitis, also streptococcal and pneumococcal meningitis. Which of these varieties of disease is present you will usually be able to determine by examining your stained film, because the meningococcus is contained within the cell, and does not stain by Gram's method, as it is a Gram-negative bacillus. The pneumococcus may or may not be within the cell, but it is Gram-positive. The streptococci are usually easily recognised. Lymphocytes, on the other hand, occur in tubercular meningitis and in spinal caries, because the meninges become infected with tubercle bacilli, and in parasyphilitic and in syphilitic diseases. Having examined the film in that way, you next make a chemical examination of the fluid. You can test for protein by boiling. Anything more than a faint haze, such as you see here, is pathological. Another important investigation is to see whether the protein is globulin or albumen. You can test for globulin by taking a saturated solution of ammonium sulphate and adding it to an equal quantity of cerebro-spinal fluid. If there is any globulin present you will see a ring formed at the junction of the two fluids. Globulin is particularly important because it occurs in parasyphilitic disease, also to a certain extent in syphilis of the central nervous system. But the presence of globulin would point to a syphilitic or parasyphilitic disease. If you have made an examination in that kind of way you will have acquired a great deal of very useful information from the fluid concerning the nature of the disease with which you are dealing. Another very important point is to apply the Wassermann test to the cerebro-spinal fluid. It is particularly useful in distinguishing between cerebro-spinal syphilis and general paralysis of the insane, which, as I have alseady said, may be very hard to distinguish clinically, and in both of which the cerebro-spinal fluid has many points

of similarity. For instance, they both have an excess of lymphocytes. Parasyphilitic diseases, especially general paralysis of the insane, is associated with a large quantity of them. Both have globulin. The Wassermann reaction, however, as a rule is negative in syphilitic and positive in parasyphilitic diseases, particularly (over 90 per cent.) in cases of general paralysis of the insane. That rule, however, is not a hard and fast one, for sometimes it is positive in syphilitic disease and sometimes negative in general paralysis. As the result of anti-syphilitic treatment the number of lymphocytes in the case of syphilis will be materially reduced, but in parasyphilitic disease it has practically no effect upon the lymphocyte count.

So much for the diagnostic point of view, which is the most important one. But I must say a few words on the therapeutic uses of lumbar puncture. Those are, first of all, under certain conditions to relieve the headache associated with increased intra-cranial pressure. Secondly, the treatment of some of the various forms of meningitis, particularly of meningococcal meningitis. The withdrawal of cerebro-spinal fluid and subsequent injection into the spinal theca of one of the various sera, Flexner's or anti-meningococcal serum, in some cases has proved of definite value. I have seen two or three cases of this post-basal meningitis which appeared to be cured by Flexner's serum in that way. Another disease that has recently been treated by intra-spinal injections is tetanus. That has been treated by intra-thecal injection of anti-tetanus serum for some time past and more recently by similar injection of magnesium sulphate. This method, introduced by Meltzer, has not been employed very largely in this country, but more so in Cairo by Phillips, who thinks the treatment of tetanus by sulphate of magnesia is more successful than by any other means. Briefly the method consists in using a 25 per cent. sterilised solution of magnesium sulphate, and injecting 1 c.c. of this within the dura for every 25 lb. body weight. I have personally seen two cases that did very well by this method. Sometimes lumbar puncture is of use in the treatment of uræmia. The patient having uræmic convulsions and being unconscious, may be temporarily benefited by this means, but I have personally very little experience of the value of lumbar puncture in this connection.

Time has only allowed me to give you a very brief outline of this interesting subject, but I have endeavoured to bring before you the most important points that arise in this connection.

January 13th, 1913.

Artificial Pneumothorax in Treatment of Pulmonary Tuberculosis.—Saugman has applied this method in the last five years in 104 cases. but found conditions in 31 preventing effectual compression of the lung. His percentage of failures is much larger than in the American reports, probably because in America the procedure is applied before the disease has reached such advanced stages. In his entire series of 2100 injections of nitrogen he has had only one fatality, and important lessons were learned from this case. The patient was a young man who dreaded the procedure. The puncture pierced only the chest-wall and no gas could be injected. Repeating the puncture it proved possible to inject 2 c.c. of the nitrogen until the manometer showed a slight negative pressure during respiration. After waiting for eight or ten minutes the cannula was introduced for the third time. When the manometer showed slight negative pressure the flow of gas was arrested. The young man suddenly collapsed, with signs of heart failure; after vomiting copiously he ceased to breathe. Stimulation was tried in vain, including massage of the heart. Necropsy failed to disclose any cause for the fatality. The findings were like those in a fatal chloroform accident. Everything pointed to reflex paralysis of the heart from pleura shock. The case teaches that the injection of gas should not be made soon after eating. and that the reflex excitability should be reduced by a preliminary injection of morphine, and, further, that not more than one puncture should be made at one sitting. Aside from this fatality, the only by-effects noted were in a few cases transient dizziness, seeing black, or distress, or brief paresis in one arm, with brief clonic contraction. In 72 of the 104 patients effectual compression was realised, and effects were obtained, he says, surpassing those possible with any other method. Of 4 patients with temperature over 39° C. 3 became entirely afebrile, 5 of 7 with temperature of 38° to 39° C., and 5 of 6 with subfebrile temperature, while 9 with occasional febrile periods have been entirely free from them since. In all of this group of 35 the tuberculous process was severe, involving the entire lung with much destruction of tissue in some, and the other lung showed some involvement in a few instances, in three of half of the lung and more. He prefers puncture to an incision, citing four cases in eighty on record in which fatal infection developed after the incision technique for pneumothorax. He thinks this positive danger far outweighs the hypothetical danger of air embolism with the puncture technique.—*Journ. A.M.A.*, vol. lix, No. 26.

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HILUS (AND PERI-HILUS) TUBERCULOSIS AND ITS SEQUELÆ.*

By R. MURRAY LESLIE, M.A., B.Sc., M.D.,
Senior Physician, Prince of Wales's General Hospital; Physician, Royal Hospital for Diseases of the Chest, London.

THE region of the hilus or root of the lung has long been recognised as a common site of tuberculous deposit, more particularly in connection with the tracheo-bronchial lymphatic glands, but the immense importance of the hilus as an early and primary focus of disease is only just beginning to be appreciated. The frequency with which the lymphatic glands situated close to the bifurcation of the trachea become affected may be readily understood when we recall the fact that these glands are the natural filters to which pass foreign particles, including tubercle bacilli, originally deposited on the bronchial mucous membrane in the act of inhalation or in the alveolar tissue of the lungs, whether originally conveyed there by aspiration or blood infection.

PULMONARY AND EXTRA-PULMONARY LYMPHATIC SYSTEM.

In order to make it clear how the virus of tuberculosis is carried to the bronchial glands and how the disease itself extends and is disseminated, one must have a right conception of the intra-pulmonary and extra-pulmonary lymphatic drainage system. It has been well said that the whole lung is pervaded in every crevice of its structure by lymphatic tissue. In the alveoli there are branched protoplasmic cells communicating with fine tubes and interstitial spaces lined by endothelial cells, while in the peri-lobular connective tissue there is a fine

* A Lecture delivered at the Medical Graduates' College and Polyclinic on October 8th, 1912.

network of tiny lymph-vessels into which the alveolar lymph passes. In the bronchial mucosa there is a network of lymphatics, the lymph capillaries being situated underneath the vascular capillaries which lie immediately beneath the bronchial epithelium. There is also a network of fine lymph-channels in the bronchial submucosa. Teichmann has shown that the lymphatics of the bronchial mucosa run parallel to the long axis of the bronchi in contra-distinction to those of the submucosa which tend to run transversely on the cartilaginous rings. From the network of bronchial lymphatics lymph-vessels pass to the tissue surrounding the pulmonary blood-vessels accompanying the bronchi. At the point where the bronchi branch we have generally three lymphatic vessels, one of which passes to the artery and the other two to the veins, while the smaller arteries and veins are only accompanied by a single lymph-vessel situated between the blood-vessel and the bronchus. We have thus a series of peri-vascular and peri-bronchial lymphatic channels into which drain the bronchial and peri-lobular lymphatics, and which end in the numerous tracheo-bronchial glands occupying the space below the bifurcation of the trachea and extending on to the pericardium, though single glands are found surrounding the main bronchi and also lying on the pulmonary artery.

Towards the surface of the lung the peri-lobular lymphatics are believed to communicate with the lymphatics of the visceral pleura. These pleural lymphatics form a network of vessels which gradually join up close to the root of the lung into four or five large lymph trunks which terminate in the bronchial glands at the hilus, where there is free anastomosis with the lung lymphatics. The existence of stomata on the surface of the visceral pleura providing direct communication between the pleural sac and the lymphatics of the lung and pleura is doubtful in the light of recent investigations.

It must be remembered also that there is *direct communication between the lymphatic system of the thorax and that of the neck and abdomen.*

As regards the neck, Grober's remarkable experiments demonstrated a direct route to the pleura and lungs *viâ* the cervical lymphatic glands, and the discovery of this route led him to believe that primary infection by way of the tonsils might be the explanation of the frequency with which the apex of the lung was found to be the seat of tuberculous disease. It is certainly fairly common to find that enlargement of the supra-clavicular and axillary glands precedes pulmonary tuberculosis. In the case of tonsillar infection the submaxillary glands are the first to be involved, the disease tending to gradually pass down to the axillary and supra-clavicular glands, and in such cases the bronchial glands are not infrequently found enlarged and caseous. The importance of this direct relationship between the cervical and mediastinal lymphatic systems may be of great importance both in regard to the incidence and spread of pulmonary tuberculosis.

As regards the abdomen, Poirier * proved the existence of free communication between the lymphatics of the pleural and peritoneal surfaces of the diaphragm. As far back as 1903, Professor Behring, of Berlin, maintained that pulmonary tuberculosis was generally the result of infection by the intestinal route, which contention received considerable support some years later from Calmette and Guerin's notable experiments with intra-œsophageal injections of bacillary emulsions in guinea-pigs.

These experiments led these observers to believe that it was the rule for the virus to be carried to the lungs by way of the abdominal lymphatic system instead of the lungs being affected directly by inhalation as had previously been supposed. I understand, however, that both Behring and Calmette have recently considerably modified their views in regard to the respective frequency of infection by inhalation and ingestion, the former being now generally regarded as the more important channel of infection.

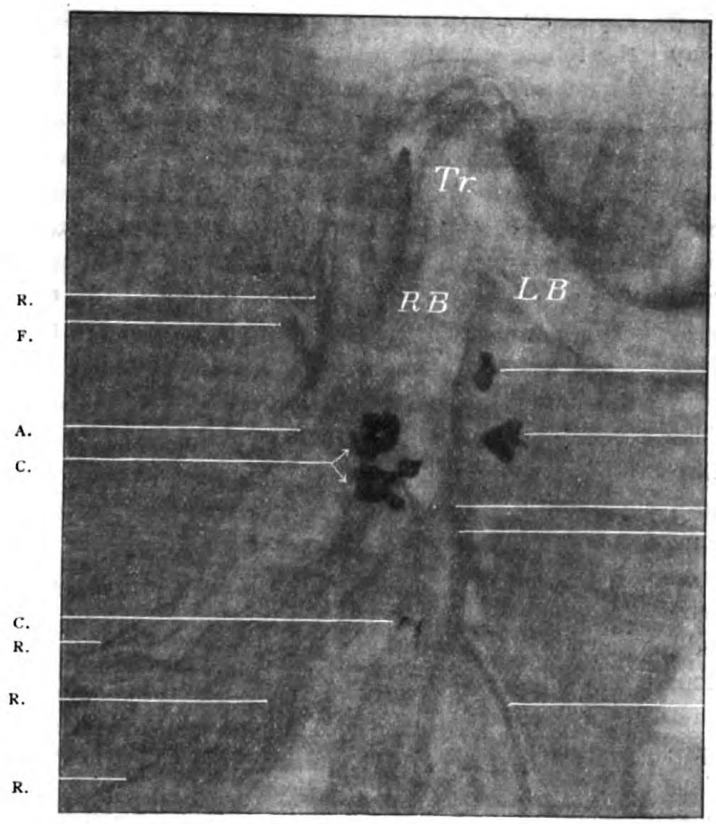
Frequency of Hilus Tuberculosis.

The importance of hilus tuberculosis is shown by the fact that the bronchial lymph-glands are often the first tissues attacked by tuberculosis in early life, and may be the only foci of disease in the whole body. So extraordinarily common are tuberculous foci in the bronchial glands and surrounding lung-tissue that the majority of all persons dying of accident or suicide in Paris, and whose bodies are exhibited at the Morgue, present

* 'Traité d'Anatomie humaine,' Paris, 1902.

evidence of old-standing or recent disease in these parts. Naegeli—the well-known pathologist—found evidence of tuberculosis in no less than 97 per cent. of all adult post-mortem examinations, and in a large proportion of these the disease took the form of calcareous or caseous deposits in the bronchial glands situated either at the roots of the lungs or along the large air-tubes. The remarkable investigations of Hamburger and Monti in Vienna indicated that over 50 per cent. of school-children under six years of age and 94 per cent. of school-children under fourteen years of age had already become infected with tuberculosis, although the natural resistance of the individual was sufficient to prevent 80 per cent. from ever exhibiting symptomatic manifestations of disease. It would probably be correct to state that in the larger number of these children the disease affected the lymphatic glands at the roots of the lungs and elsewhere.

It is, indeed, at the period of infancy and early childhood that the tuberculous seed is commonly sown in the organism, although the disease may remain latent for a prolonged period and only manifest itself when the system is lowered by some acute illness. Professor Nietner, of Berlin, who has specially devoted himself to the study of tuberculosis in childhood, is of opinion that tuberculous deposit often commences in the middle of the second year of life and continues in an



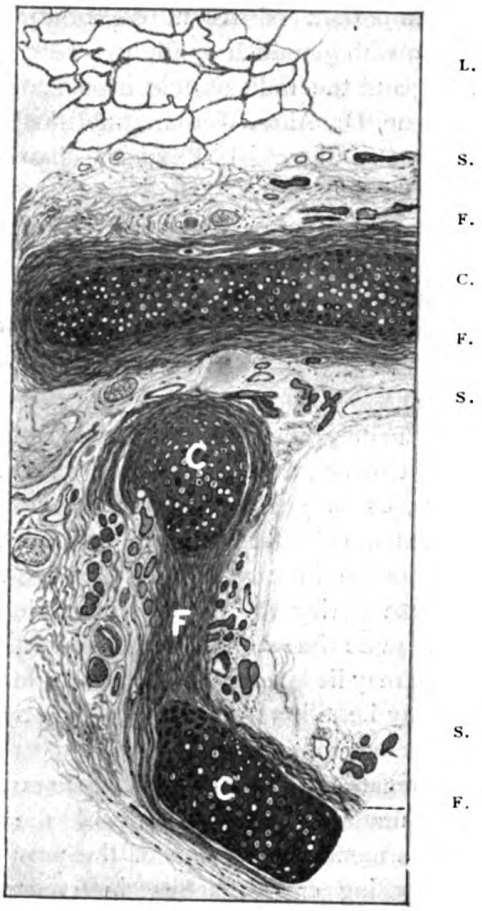


FIG. 1.—Skiagram of healthy lung from post-mortem room. C. Calcareous patches in bronchial glands. A. Air-tubes surrounded by fibrous tissue. F. Thick fibrous sheath round air-tube. R. Radiating shadows due to fibrous tissue; round smaller air-tubes. Tr. Trachea. R.B. Right bronchus. L.B. Left bronchus. (By kind permission of Dr. Jordan.)

FIG. 2.—Microscopical section through same lung shown in Fig. 1 through point S, showing great increase of fibrous tissue around bronchial cartilage (cartilage being transparent to X rays). L. Healthy lung. S. Stroma. C. Bronchial cartilage. F. Fibrous-tissue sheath (tuberculous giant-celled systems sometimes present). (By kind permission of Dr. Jordan.)

increasing ratio every year up to and beyond the age of puberty. Similar observations in regard to the prevalence of tuberculosis in children have also been made by Dr. Phillip, of Edinburgh, who found actual stigmata of pulmonary tuberculosis in no less than one third of the children attending the schools of that city.

The work of Dr. Anton Ghon,* of Prague, on pulmonary tuberculosis in children is one of the most important of recent contributions in connection with glandular and hilus tuberculosis, while in England the radiographic investigations of my colleague, Dr. Alfred Jordan, published under the title "Peri-bronchial Phthisis" have attracted wide-spread attention. Dr. Jordan states that the great majority of a series of "healthy" lungs obtained by him from the post-mortem room and examined by the X rays showed calcareous deposits in the lymphatic glands, either at the roots or along the course of some of the larger air-tubes (Fig. 1).

Fortunately there is a marked tendency to spontaneous cure in the case of glandular tuberculosis, as is evidenced by the frequency with which such calcified bronchial glands are met with post mortem in cases where there had been no suspicion during life of any tuberculous disease. At the same time it must not be forgotten that the disease may lie latent in the glands for long periods, and may be followed many years later by important and serious consequences.

As regards the incidence of the disease, evidence is accumulating that catarrh of the bronchial mucous membrane is one of the most important predisposing causal factors in connection with hilus tuberculosis. Sir William Osler in this way explains the frequent occurrence of tuberculous adenitis of the bronchial glands as a sequela of whooping-cough and measles. Increased vulnerability of the tissues, according to the late Professor Virchow, is the important factor in producing the disease, and such vulnerability would naturally occur as the result of injury or breach of continuity in connection with the bronchial mucosa.

It is also more than probable that when bronchitis has persisted for some time and the ciliated epithelium has thereby become damaged and its functions impaired, bacilli may then find their way by aspiration into the remote bronchial ramifications and may be carried into the alveoli of the lung. Thence they pass between the cells lining the air-vesicles and so into the lymph-channels and connective-tissue spaces of the alveolar walls.

From the frequency with which enlarged bronchial glands and tuberculous thickening in the adjacent pulmonary tissues are found in persons exposed to dust inhalation, it is conceivable that the tubercle bacilli may in many cases reach the bronchial glands by being actually attached to the organic particles, which would thus act as the direct carriers of the infective virus. At the same time the association of silicosis with tuberculosis may also be explained by the theory that inorganic particles—particularly when of a sharp, angular character—may cause sufficient irritation of the glandular and pulmonary tissues to render the resulting inflammatory area a suitable nidus for the growth and development of the bacillus of tuberculosis.

The Primary Site of Chronic Pulmonary Tuberculosis: Apex *v.* Hilus.

Hitherto the apex has always been regarded as the most frequent primary focus of pulmonary tuberculosis. In 1880 Sir James Kingston Fowler* stated that a spot situated from one to one and a half inches below the actual apex was the primary seat of disease in the large majority of cases. This observer also drew attention to the fact that tuberculous disease in its progress through the lungs followed a distinct "line of march" particularly in the chronic and fibroid varieties, the disease spreading in each lobe from above downwards, hardly ever from below upwards. From the primary apical focus he found that the lesion tended to extend in the first instance backwards and then downwards along the anterior portion of the upper lobe in the form of scattered nodules situated about three quarters of an inch within its margin. The apex of the lower lobe became affected at a comparatively early period, the disease tending to spread backwards along the posterior surface of the lung and laterally along the line of the inter-lobar septum. When extension took place along the

* 'Der Primare Lungenherd bei der Tuberculose der Kinder,' Prague, 1912.

* 'Localisation of Lesions in Phthisis,' 1880.

base it was usually by means of scattered nodules of infiltration, often arranged in a racemose manner. Soon after the apex of the lower lobe was affected there was an involvement of the apex of the upper lobe of the opposite lung, to be followed later by infiltration of the apex of the lower lobe of that lung.

It is important, however, to observe that Kingston Fowler mentions occasional variations in the position of the primary foci and also in the "line of march," and makes special note of so-called cross lesions where the extension seems to take place from the apex of the upper lobe of one lung to the apex of the lower lobe of the other. He also makes special reference to the comparative freedom of the bases of the lungs from tuberculous deposit, stating that primary basic disease is exceedingly rare, there being generally evidence of older lesions of the apices. Fowler attributes the frequency of apical disease to the lesser functional activity of this portion of the lung, which is imperfectly expanded in ordinary quiet breathing, such impairment of function being associated with defective resisting power. It must be admitted, however, that Fowler makes no attempt whatever to explain the rationale of the "line of march" which he so carefully describes. It would seem that a more or less constant "line of march" must have some rational explanation.

On the other hand, Dr. Alfred Jordan* states categorically that chronic pulmonary tuberculosis does not generally commence at the apex, but begins at the roots and extends along the branchings of the large bronchial tubes. I am indebted to the courtesy of Dr. Jordan for permission to reproduce Figs. 1 and 2, already published in the 'Practitioner,' representing calcified glands and peri-bronchial fibrosis.

As regard the mode of infection, and the manner in which the virus gets to the peri-bronchial tissue in the first place, Dr. Jordan does not commit himself to any definite opinion, nor has he given any explanation as to why the peri-bronchial pneumonic process should commence around the large bronchi close to the root of the lung instead of more or less promiscuously around the smaller and more peripheral bronchi.

Dr. Jordan inclines to the view that the primary tuberculous deposit does not take place in the bronchial glands, but in the peri-bronchial tissues in the form of broncho-pneumonic foci, and that the glandular enlargement is secondary.

In a number of cases the infection seems to spread directly to the lungs from the caseous bronchial glands, often apparently by direct contact. Indeed, as Sir William Osler has pointed out, and his statement is confirmed by my own observations, it is sometimes difficult to determine in a section through the affected lung where the caseous bronchial glands terminate and where the pulmonary tissue begins. In the greater number of cases, however, the extension is probably by way of the lymphatic network already described as surrounding the pulmonary vessels and bronchi. It is possible that the extension takes place in a great measure along the peri-vascular lymphatics surrounding the pulmonary vessels, but owing to the close proximity of the pulmonary vessels and bronchi the task of precise discrimination between the peri-vascular and peri-bronchial lymphatics is naturally difficult. It may be that while the larger number of bacilli entering by way of the bronchial mucosa are passed on to the bronchial glands, into which the bronchial, peri-vascular and peri-bronchial lymphatics drain, a certain number of bacilli filter through the mucous, submucous and peri-bronchial lymphatics, and may give rise respectively to tubercles on the surface of the bronchial mucosa, to caseous deposits in the bronchial submucosa, and to broncho-pneumonic areas in the pulmonary tissue surrounding the bronchi. Dr. Jordan favours the view that the tuberculous process generally spreads in the form of broncho-pneumonic extension in the peri-bronchial pulmonary tissues, which in favourable cases may cicatrise into fibrous bands, appearing as dark radiating shadows on X-ray examination of the affected lungs.

Dr. Jordan states that in 40 per cent. of cases of phthisis the disease commences as a definite peri-bronchial infiltration and spreads from the hilus in all directions, but most rapidly along the ascending and descending branches of the main bronchus, and that sooner or later the disease reaches the apex by way of the ascending bronchiole. When the apex is reached, the disease, he says, may there progress rapidly, so

* 'The Practitioner,' February, 1912; 'Brit. Med. Journ., August, 1912.

that the apex appears clinically to be the chief seat of invasion, the primary disease at the root being so deeply placed as to escape detection from without. He explains purely apical cases on the hypothesis that the tuberculous infection has travelled up from the hilus along the main ascending bronchiole to the apex in an early stage, and progresses there, while the track of infection from the root to the apex has healed up, leaving only an excess of fibrous tissue along the ascending bronchiole. Dr. Jordan bases his conclusions mainly on his interpretations of radiographic shadows in the lungs of living subjects, and of similar shadows obtained by the X-ray examinations of lungs taken from the post-mortem room. Now, ingenious and striking as these theories and conclusions are, it is doubtful if Dr. Jordan has so far produced sufficient evidence to warrant definite dogmatic generalisations.

In any case we have here two divergent views—the one ably expounded by Kingston Fowler as the outcome of unrivalled pathological and clinical experience to the effect that pulmonary tuberculosis is primarily an apical disease with gradual extension downwards along a definite "line of march," while the newer view is advanced by an expert radiographer with much less pathological and clinical knowledge, who nevertheless has the courage of his convictions, and boldly states his belief that apical tuberculosis is but an extension of "peribronchial phthisis" which is really the primary manifestation of chronic pulmonary tuberculosis. Is it possible that two views apparently so divergent can be reconciled?

Kingston Fowler* himself, in discussing the pathology of chronic fibroid phthisis, admits that the extension of the disease is more towards the peri-bronchial tissue than towards the alveoli, and states that the bronchi may be thickened from small-celled infiltration, while actual tubercles may be present both in the walls of the bronchi and in the peri-bronchial tissue, in which cases groups of granulations having a racemose arrangement are usually present (see Fig. 3). Fowler also found that the lining membrane of bronchi which pass through such tuberculous broncho-pneumonic areas are often intensely injected and swollen, and may even have undergone extensive tuberculous ulceration. He further found that tuberculous infiltration of the peri-bronchial sheath may lead to marked thickening of the tube and narrowing of its lumen. In some of these cases this fibrous constriction and narrowing of the bronchus may lead to bronchiectatic dilatation beyond the site of constriction.

As far as Dr. Jordan's views are concerned, he does not seem to have fully appreciated, and, indeed, has practically ignored, the frequent occurrence from time to time of autogenous blood infection, aspiration broncho-pneumonia and other accidental contingencies, not to mention the possibility of actual primary alveolar deposit as the result of inhalation. He has disregarded to a great extent the consideration of what the Germans describe as "open" tuberculosis. "Open" pulmonary tuberculosis includes the type in which fragments of muco-pus loaded with autogenous

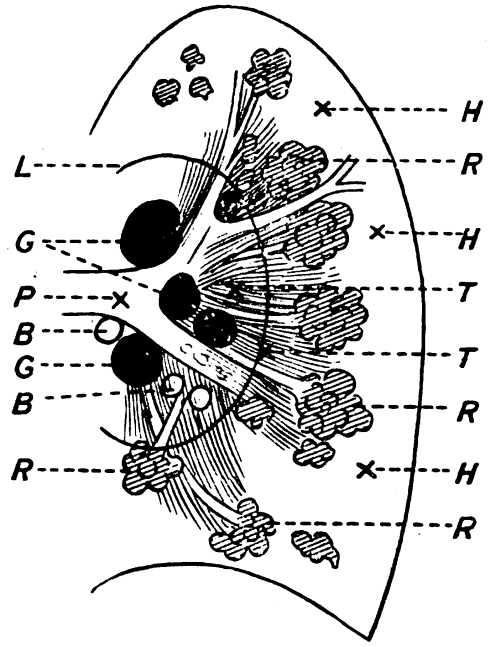


FIG. 3.—Chronic fibroid phthisis commencing as hilus tuberculosis. This drawing was made from a specimen in Brompton Hospital Museum (Catalogue No. 116) and represents a complete section through right lung. Is an excellent example of chronic fibro-caseous phthisis, the disease having evidently commenced at the hilus and extended outwards into the surrounding tissue in a wonderfully symmetrical fashion. P. Pulmonary vessel cut longitudinally. B. Air-tubes cut across. G. Caseous bronchial glands at hilus. T. Tuberculous fibrous thickening and infiltration extending outwards from hilus. R. Racemose tubercle in peri-hilus area. L. Imaginary line circumscribing hilus of lung. H. Peripheral zone of healthy lung-tissue ¾ in. wide. (Drawing by author.)

* 'Diseases of the Lungs,' Fowler and Godlee.

bacilli may pass directly into the lumen of the bronchi, thus forming what might be described as tuberculous "bronchial emboli," capable of being aspirated into the terminal bronchioles and alveoli as well as of being expectorated in the sputum. Dr. Jordan's views regarding the universality of peri-bronchial extension might be quite sound if he were dealing only with a "closed" disease with no bacilli in the bronchial contents and spreading along the lymphatic lines of least resistance, but there is in actual fact a constant liability to accidental contingencies. On the one hand we may have the erosion of a blood-vessel by a tuberculous gland or other diseased focus, so that the virus is carried by the blood-stream and deposited at the less resistant apices or elsewhere, while on the other hand we have the above-mentioned tuberculous "bronchial emboli" which may be carried by inspiratory efforts to the apices and peripheral portions of the lungs possibly at a comparatively early stage of the disease, just as occasionally happens in the case of the classical "phthisis ab hæmoptoe" when new deposits occur after a pulmonary hæmorrhage due to the aspiration of the infected blood into distant portions of the pulmonary tissue.

The presence of autogenous bacilli at an early stage in the bronchial secretions may indicate one of several processes. There may be small miliary tubercles in the bronchial mucous membrane, which, on breaking down, form small tuberculous erosions discharging tubercle bacilli into the bronchi; there may also be present large caseous nodules in the submucous tissue which may burst into the lumen of the tube; and more important still in the course of the tuberculous broncho-pneumonic extension above referred to, it is quite common to find in addition to and *pari passu* with the cicatrising fibrous process already described, that some of the broncho-pneumonic patches may caseate, not a few breaking down into minute cavities communicating more or less freely with the adjacent bronchioles, into which the tuberculous contents of the alveoli escape.

In any or all of these contingencies which are so common in the later stages of the disease to as practically constitute an essential part of the pulmonary tuberculous process, we have all the conditions necessary to bring about an aspiration broncho-pneumonia. Fragments of muco-pus loaded with bacilli may be sucked in with inspiration and carried to the apices and other portions of the lungs, where they may lodge and set up tuberculous inflammatory mischief. The ciliated epithelium is no longer able to expel such fragments, and the phagocytic action of the lining cells of the bronchi, bronchioles and alveoli, though generally quite effective in the case of the few individual air-borne bacilli carried from without by inhalation, may be no longer capable of dealing with such concentrated masses of bacilli, accompanied as they are by degenerated epithelial *débris*, which may itself act as an irritant in addition to furnishing pabulum for the accompanying bacilli, until they succeed in obtaining a fresh nidus in the pulmonary tissue suitable for their growth and development. Areas of tuberculous broncho-pneumonia are thus formed as independent foci, and these are specially apt to occur at the apices of the lobes, where there is least functional activity and consequently least power of resistance.

The frequency of the pulmonary apex as the seat of tuberculous disease may be explained on the supposition that its normal inactive condition in the course of normal respiration induces a certain degree of local anæmia (ischæmia). The result is that we have the very conditions most likely to favour the deposit and development of the tubercle bacillus, such deposit not being surrounded by well-filled blood-capillaries which are uniformly present in the actively functioning areas of the lung at the base and elsewhere. At the apex the deposit is inadequately bathed by blood-serum containing protective and anti-bactericidal properties; there is, in short, as Sir Almroth Wright would express it, "defective lymph lavage," thus converting the apical focus into a "non-bacteriotropic nidus."

The advocates of the view that the hilus is generally the primary focus of pulmonary disease find it difficult to determine whether apical infiltration occurs independently of *Hilus tuberculosis* as patients seldom die at the early stage, unless from some extraneous cause. In the case of patients who die with advanced disease of one lung, and are found post mortem to have early apical deposit in the opposite lung, there are always hilus deposits, and therefore such cases throw but little light on the problem.

The principal evidence in support of the hilus as

a primary focus of disease is obtained from radiographic examinations of early cases, and this evidence is by no means absolutely conclusive. The difficulty arises from the fact that in the lungs of nearly all presumably healthy persons we find opaque areas at the roots situated just outside the cardiac shadow, while linear radiating shadows —the so-called "hilus shadows"—pass out into the lung substance for varying distances. There is at present considerable difference of opinion in regard to the significance of these hilus shadows. Some of the German radiographers, indeed, regard these radiating shadows as representing the normal pulmonary vessels filled with blood, while other radiographers are equally positive that they are definitely pathological. Thus Dr. Jordan states that they are due, in the majority of cases, to the previous entry of tubercle bacilli and to the fibrosis resulting from the reaction of the organism to render these bacilli inert.

Hilus shadows and blotches and radiating striations of varying density confined to the roots are practically universal, and invariably present to a greater or less extent in all adults. Accordingly experienced radiographers, such as Dr. Ironside Bruce and Dr. Jordan, are of opinion that *per se* they have but little significance in adults except as being indicative of old inflammatory deposit—tuberculous or otherwise.

The fact that Naegeli and other pathologists found evidence of tuberculous deposit almost constantly at the roots of all lungs examined by them post mortem certainly lends support to such a theory, though it does not actually prove the correctness of the interpretation of the X-ray hilus shadows. In any case, as long as the shadows are confined to the root area, it is at present extremely difficult to differentiate between old-standing probably cured disease contracted in childhood and recent tuberculous infiltration. In the majority of adult cases exhibiting shadows strictly confined to the roots it is safe to conclude that if they are pathological they indicate merely old-standing cured disease contracted in childhood and not recent tuberculous infiltration.

Another difficulty arises from the fact that in long-standing bronchitis, bronchiectasis and silicosis one finds similar, though possibly coarser, radiating shadows, which may be the result of cicatrised inflammatory areas due to non-tuberculous irritation (Fig. 10). Here, again, however, it is difficult to exclude tuberculous infiltration as a secondary process, which is undoubtedly a common accompaniment in gold-miners' and tin-miners' phthisis and other forms of pneumokoniosis. If in doubt it is often well, in the absence of tubercle bacilli in the sputum, to have recourse to the subcutaneous tuberculin test. Well-marked hilus shadows, particularly if they extend into the lung substance well outside the root area in conjunction with a positive tuberculin test, I believe to be extremely significant of tuberculous disease.

There are, however, other important X-ray appearances which are of great confirmatory value. It has long been known that restricted movement of the diaphragm, as seen through the fluorescent screen, is frequently present in early pulmonary tuberculosis, such restricted movement occurring before any definite shadow at the apex or other portion of the lung tissue proper becomes evident. May it not be that this restriction of movement may, in reality, be due to hilus disease (glandular, or pulmonary, or both) restricting the air entry?

A vertically placed tube-like heart is another confirmatory sign, and is supposed to be characteristic of tuberculous soil and to be one of the dystrophies which predisposes to tuberculosis. When the heart occupies its normal oblique position the root opacities are not of such importance.

Any X-ray evidence of tuberculous deposit outside the root area is of course highly significant. One might specially mention the imperfect lighting-up with inspiration of individual areas of the lung-tissue on examination with the fluorescent screen; also grape-like clusters of minute opacities occurring outside the root area which generally indicate foci of tuberculous broncho-pneumonia (see Fig. 4).

It is difficult to draw a hard and fast line of demarcation between the hilus proper and the pulmonary tissue outside the hilus—the peri-hilus. When viewed antero-posteriorly a vertical line drawn through the costo-chondral articulations affords a fairly accurate dividing line for ordinary clinical purposes (see Figs. 4, 6, 9, 10).

Incidentally it may be mentioned that in taking a radiograph of the lungs it is most important that the patient should hold his breath during the time of exposure to the X-rays.

In examining for enlarged glands in the region

of the hilus a lateral oblique examination should always be made, as this frequently reveals a considerable enlargement of these glands, which otherwise would escape detection as they are often concealed by the normal median shadow when examined antero-posteriorly. If much enlarged, they may cause a marked increase of the normal median opaque area (produced by the sternum, aorta and upper dorsal spine), and this broadening of the mediastinal shadow may be so pronounced as to simulate aortic aneurysm. The presence of very dark root opacities indicating calcified foci definitely proves the existence of tubercle in a quiescent or cured condition, and is strong evidence that the

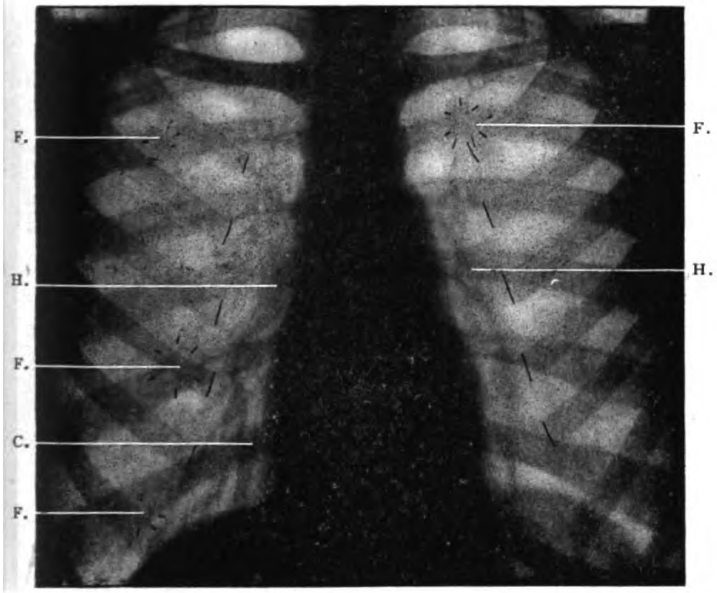


FIG. 4.—Skiagram of chest from supposed case of spasmodic asthma following whooping-cough in boy, æt. 9 years—in reality a case of hilus tuberculosis. Note blotching (H) at roots of both lungs (most distinct on right side) lying external and parallel to the narrow vertically placed cardiac shadow. At the right hilus there are two small opaque spots suggesting old calcified foci (C). Linear shadows may be traced passing towards the periphery and extending in the direction of the apices as high as the level of the second rib in front. Here and there throughout both lungs may be seen small rounded opacities (F) arranged in clusters, indicating foci of tuberculous broncho-pneumonia. *N.B.* — After three months' open-air treatment at Bournemouth patient gained one stone in weight, and the asthmatic attacks entirely disappeared. (Radiogram by Dr. Ironside Bruce.)

hilus root shadows are probably also tuberculous in origin (see Fig. 5).

It goes without saying that the well-known mottling and dark shadows so characteristic of active tuberculous disease in a more advanced

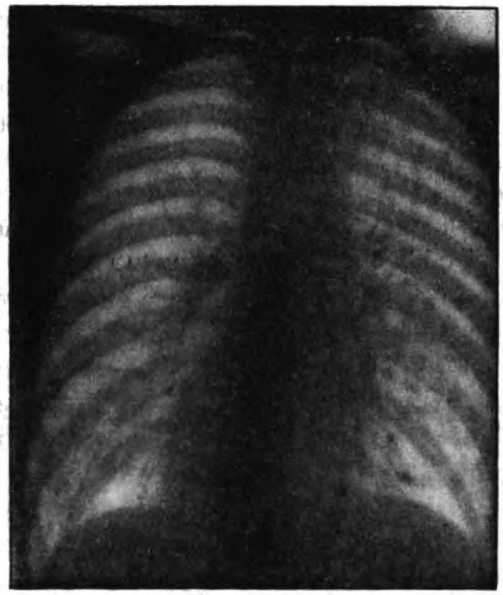


FIG. 5.—Skiagram of chest of a boy, æt. 11 years, admitted to Royal Chest Hospital with obscure symptoms suggestive of pulmonary tuberculosis, but no definite physical signs. There are numerous calcified foci in the hilus and peri-hilus areas on the right side, apparently situated along the ramifications of the air-tubes and pulmonary vessels. Linear shadows may be seen on both sides passing out from the root blotches in the direction of the right apex and left base. (Radiogram by Dr. Jordan.)

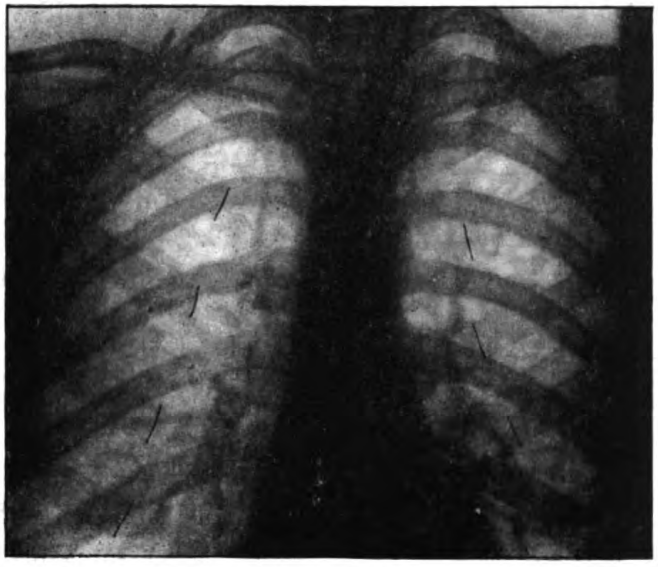


FIG. 6.—Skiagram of hilus tuberculosis (supposed bronchial catarrh). From case of gentleman, æt. 24 years, with three months' history of obstinate cough and slight loss of weight, but no physical signs except deficient expansion of chest. Note the well-marked hilus blotches, some of which may represent calcified glands, and also the linear striations passing upwards and outwards from the root area. There was defective movement of diaphragm. The hilus shadows indicate old healed tuberculous deposit, while the small rounded opacities observable here and there are probably due to more recent tuberculous foci. (Radiogram by Dr. Ironside Bruce.)

stage would be even still more confirmatory, but here we are dealing mainly with pulmonary tuberculosis in its earlier manifestations, before the various accidental contingencies of autogenous infection and aspiration broncho-pneumonia have occurred.

Apart, however, from X-ray evidence, there is a considerable and growing amount of pathological evidence in support of hilus tuberculosis as an early manifestation of pulmonary disease. It is quite common to meet post-mortem with cases of fairly

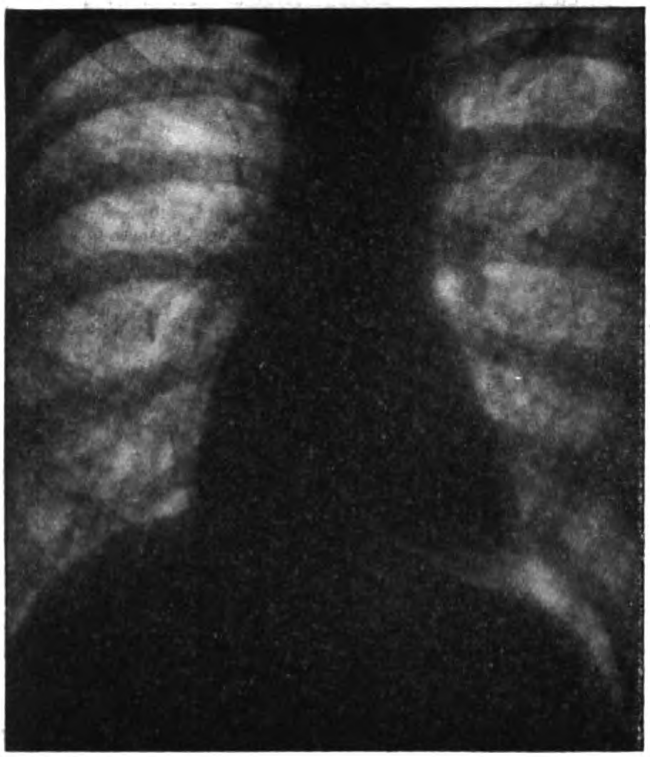


Fig. 7.—Skiagram from lady, æt. 44 years, with history of recurrent hæmoptysis, but no cough and no definite physical signs, except deficient air-entry at right apex. Well-marked reaction with 5 mgrm. of old tuberculin. Appearances indicate old hilus tuberculosis. Note the hilus blotches with coarse linear shadows passing towards apices and right base, indicating fibrous thickening along the air-tubes. The dark rounded root opacities probably represent enlarged bronchial glands. (Radiogram by Dr. R. Knox.)

extensive disease more or less limited to the root area and to the lung tissue immediately surrounding the root area, while the apices and peripheral portions of the lungs may be wholly or comparatively free from tuberculous infiltration. Fig. 3 is a good illustration of this type of case, of which I have seen a considerable number, in which extensive central disease is surrounded by a peripheral shell of healthy pulmonary tissue.

I have already referred at length to the almost universal presence in all post-mortem examinations of tuberculous deposit in the tracheo-bronchial glands and surrounding pulmonary tissue in the neighbourhood of the hilus.

The Retro-Impulsion of Lymph as an Explanation of Hilus Tuberculosis.

The *retro-impulsion of lymph* is now a well-proved phenomenon, the back-flow taking place

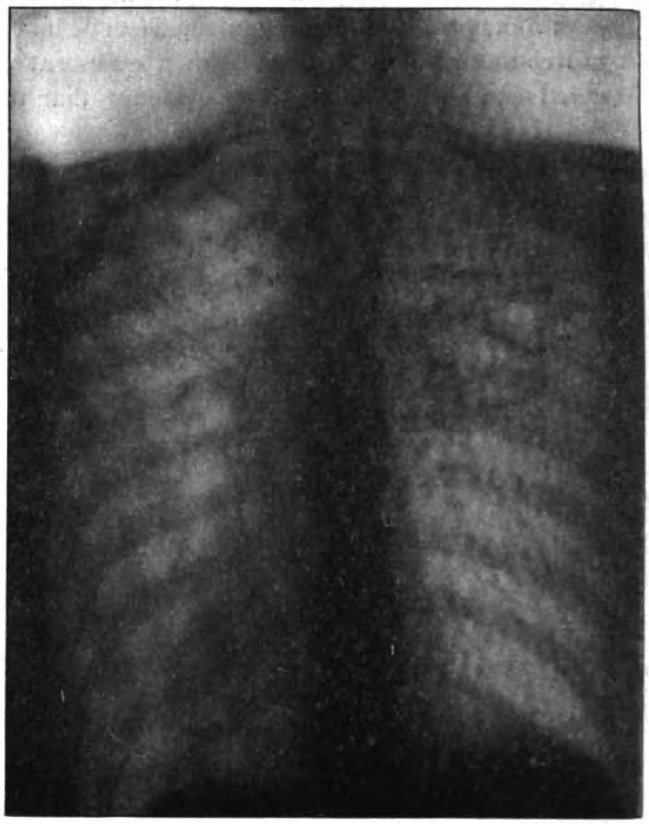


Fig. 8.—Skiagram of chest of young lady, æt. 32 years, showing spread of tuberculosis from hilus to apices, particularly on the right side, where the mottled areas may be seen filling up the space between the hilus shadow and the apex. Note also the elongated tube-like heart. Notwithstanding the history of a slight cough for two years, and the presence of tubercle bacilli in the sputum and the X-ray evidence of extensive disease of the right upper lobe, patient looked wonderfully well, and the physical signs were mainly confined to the right inter-scapular region, where there was slight dulness, prolonged expiration, and bronchophony. (Radiogram by Dr. Alfred Jordan.)

along the distended lymph-channels, the valves of which are no longer competent. Sir Almroth Wright has observed this backward flow of lymph as being of frequent occurrence in bubonic plague —the extension taking place against the usual

course of the lymph-stream. The same phenomenon may be observed in the case of tuberculous cervical glands in childhood, the extension of the disease occurring in the opposite direction to the lymph flow. Experimentally it is comparatively easy to inject such a substance as adrenalin into the subcutaneous tissue of a limb and make it pass from the proximal to the distal portion of the lymph channel. This, indeed, is the principle of Bier's treatment of distal disease by a proximal bandage of a limb, and is also the rationale of Sir Almroth Wright's back-massage in the treatment and cure by lymph lavage of tuberculous and inflammatory conditions of the terminal portions of the extremities.

Let us now apply the doctrine of retro-impulsion of lymph to explain the incidence of hilus tuberculosis.

That there is naturally a special predisposition to tuberculous deposit in the region of the hilus of the lung will appear from the following considerations:

(1) The great proportion of the foreign particles in the inspired air, including tubercle bacilli, impinge and lodge on the mucous surface of the trachea and larger bronchi. The bacilli, once they come in contact with the moist bronchial mucosa, are thereby imprisoned and their further progress along the bronchial tree is arrested. Many of these are killed by the phagocytes, while a few may escape. These are absorbed by the pigment-cells of the mucosa, or find their way through some breach of continuity and pass on by way of the bronchial and peri-bronchial network of lymphatics towards the hilus in the direction of the lymph-stream.

Further, all the living bacilli which have escaped the phagocytic action of the cells of the bronchial mucosa and connective-tissue stroma, in addition to those which may have been derived from the cervical and abdominal lymphatic systems, become eventually concentrated in the region of the root, containing, as it does, the terminal portions of the lymphatic drainage system of the thorax.

(2) After a time—it may be a period of years lasting from infancy to puberty—the bronchial lymph-glands which are the ultimate destination of the bacilli and other foreign particles may become caseous and eventually calcareous, so that the lymph-stream is blocked and the afferent lymph-vessel walls become distended, particularly at the terminal portions, so that the valves are no longer competent and a slow backward flow of lymph sets in.

(3) As a result the tubercle bacilli present in the sluggish backward-flowing lymph-stream filter through the walls of the distended lymph-channels and set up a broncho-pneumonic process in the surrounding pulmonary tissue. As these inflammatory areas heal up and cicatrise, the terminal lymph-vessels become still further obstructed by the cicatricial tissue, and so the filtering, inflammatory, and healing process is repeated a little further out, the extension taking place slowly and steadily from the root towards the periphery and mainly in the tissues surrounding the bronchi and pulmonary vessels.

(4) The whole inhalation process is as a general rule so extremely slow (in the absence of accidental contingencies producing autogenous infection) that there is as a rule plenty of time for protective antibodies to be formed in the system and for immunity to be gradually established. In this way the hilus tuberculous infiltration gradually entirely heals and cicatrises, while the increased phagocytic efficiency of the bronchial mucosa prevents the access of fresh living bacilli. This affords a rational explanation of the fact that although according to Hamburger and Monti 94 per cent. of the population under fourteen years of age have actually been infected with tuberculosis, fully 80 per cent. of these never exhibit symptoms or signs of manifest disease.

The retro-impulsion of lymph theory, of course, presupposes the hypothesis that the glands are the primary seats of tuberculous deposit, which is no doubt true in a large proportion of cases. It is probably equally true that in a certain number of instances broncho-pneumonic extension in the peri-bronchial and peri-vascular pulmonary tissue is a primary phenomenon and the result of direct infection from the bronchial mucous membrane with secondary involvement of the glands, the extension in such cases taking place in the direction of the lymph flow, the glands acting as the natural barriers in preventing systemic infection.

Confirmatory evidence of the importance of tuberculosis spreading from the hilus into the lung-tissue is obtained from a study of the various forms of pneumonokonioses, such as anthracosis, silicosis, etc. In the case of anthracosis (coalminers

disease) the carbon particles are found deposited mainly in the bronchial glands, while fibroid changes usually begin in the peri-bronchial lymph-tissue, and, indeed, in the early stage of anthracosis the fibrotic changes are almost entirely confined to these parts. We get limited patches of fibroid broncho-pneumonia, which here and there may coalesce into greyish-black areas of fibrosis. Cornet states that similar conditions govern the inhalation of tubercle bacilli and that the greater number of the inspired bacilli are deposited on the ciliated epithelium of the upper passages and tend to be swept outwards by the current of mucus, though some find their way through the mucous membrane, and are carried by the lymph-stream to the bronchial glands, which occurs with special frequency in the case of children.

The rapidity of the extension of the tuberculous process in the lungs is as a rule inversely proportional to the degree of immunity—natural or acquired—the determining factors being not so much the numbers or virulence of the invading bacilli, as the resistive power of the invaded tissues on the one hand, and the degree of vulnerability and phagocytic efficiency of the bronchial mucous membrane on the other. This, indeed, is the probable explanation of the difficulty of producing tuberculosis of the lungs in guinea-pigs by the experimental introduction of bacillary emulsions into the trachea.

The fact that most of us have had at one time tuberculous deposit now represented by some fibrous thickening at the roots may in reality be an advantage. A definite degree of immunity has thus been established, and we are actually in a better condition to resist any future invasion than if we had never contracted the disease at all. It is probable, indeed, that the gradual reduction in tuberculosis mortality is largely due to the fact that the population is gradually becoming immunised in this way. This theory will serve to explain the alarming spread of, and mortality from, tuberculosis among the native populations of Bengal, the West Indies, Australia, and elsewhere, who have not been immunised.

The resistive power of the tissues depends mainly upon heredity, but also upon dietetic and other environmental conditions, while the vulnerability of the bronchial mucous membrane, as already mentioned, is greatly increased by catarrhal conditions such as are so frequently met with as accompaniments of measles and whooping-cough, which necessarily impair its integrity.

Sequelæ of Hilus Tuberculosis.

(1) *Chronic fibroid phthisis.*—Though there is in the large majority of cases of hilus tuberculosis a tendency to a natural cure of the malady, it is far otherwise in the minority of instances which ultimately exhibit symptoms and physical signs of manifest tuberculous disease. In a certain proportion of cases, probably as the result of individual lack of resistance, hereditary or acquired, the broncho-pneumonic areas, instead of healing up and cicatrising, continue to spread and coalesce with other similar foci to form larger patches of racemose tuberculous broncho-pneumonia. These, though undergoing a certain amount of fibrotic transformation, slowly but steadily advance until eventually there is produced the familiar condition known as chronic fibroid phthisis, which in some cases may reach a fairly advanced stage before it can be detected by the ordinary clinical methods of physical examination owing to the fact that the disease may not have reached the peripheral portions of the lung, which are alone accessible to the clinical observer through the medium of palpation, percussion, or auscultation.

Fig. 3 is a drawing of a specimen in the Brompton Hospital Museum from the case of a man, æt. 34 years, who died in the hospital. The following is the description of the naked-eye characters of the right lung taken from the Museum Catalogue published in 1904: "Numerous recent racemose tubercles with pigmented centres and caseating circumferences are seen *affecting the central portions of the upper and lower lobes, a shell of practically unaffected lung-tissue about two thirds of an inch thick separating them from the pleural surface.* This specimen explains how in some cases extensive tuberculous disease of the lungs may be present and yet the physical signs may be scanty or altogether absent."

In many cases, however, long before this advanced stage is reached, there is physical evidence of disease at the apex or elsewhere (Figs. 7, 8, and 9).

Whether it is true, as Dr. Jordan maintains, that the tuberculous infection often travels in the

peri-bronchial tissue from the hilus to the apex at an early stage, the disease progressing at the apex while the track of infection from the root heals by a process of fibrosis, has not yet been confirmed by other observers. I think this apical complication is quite as likely, at any rate in a considerable portion of cases, to be due to some accidental contingency producing autogenous infection, whether by way of the blood-stream or bronchial channels, or possibly even by direct exogenous infection through inhalation.

The view that apical tuberculosis is *always* secondary to hilus tuberculosis spreading upwards by means of peri-bronchial extension cannot be maintained, but that such a sequence occasionally occurs at a comparatively early stage is more than probable.

In Fig. 3 the disease seems to have extended more or less continuously in all directions, though the principal extensions are along the pulmonary vessels and air-tubes.

(2) *Tuberculous pleurisy.*—The great majority of subacute and chronic tuberculous pleurisies, whether dry or accompanied by sero-fibrinous effusion, are tuberculous in origin, although there may be nothing in the lungs or elsewhere to suggest the presence of tuberculosis. Sir William Osler states that in almost every instance of tuberculous pleurisy there are really tuberculous foci in the bronchial glands or lung-tissue. I have already mentioned that the pleural lymphatics join up into four or five large lymph-trunks, which terminate in the bronchial glands at the hilus. Applying here also the doctrine of the retro-impulsion of lymph, the ready extension of the tuberculous process from the hilus to the pleura is obvious. The extensive tuberculosis of the pleuræ and the mediastinal glands so frequently found in cattle is significant in this connection.

I have submitted many patients with tuberculous pleurisy to X-ray examination, and in most cases there was evidence of hilus infiltration.

(3) *Asthma.*—In a large proportion of cases asthma *in children* is really due to reflex or direct irritation from enlarged bronchial glands and peri-bronchial thickening and infiltration. I have lately had several children suffering from asthmatic attacks examined by X rays, and almost invariably we have found unusually well-marked hilus shadows, and not infrequently dark discrete areas indicating calcified glands. Fig. 4 is an excellent illustration of such a case. In some cases large glandular masses have been discovered, particularly when viewed from the right and left lateral aspects. Occasionally the thoracic nerves may become involved in the hilus thickening, particularly the recurrent laryngeal branch of the vagus nerve, and such nerve-compression may have an important influence in the production of the asthmatic attacks, although in children of a nervous diathesis the mere irritation and compression of the bronchial tubes may of itself be sufficient to induce asthma. In adults also asthmatic attacks are undoubtedly in some instances associated with hilus tuberculous thickening (Fig. 9).

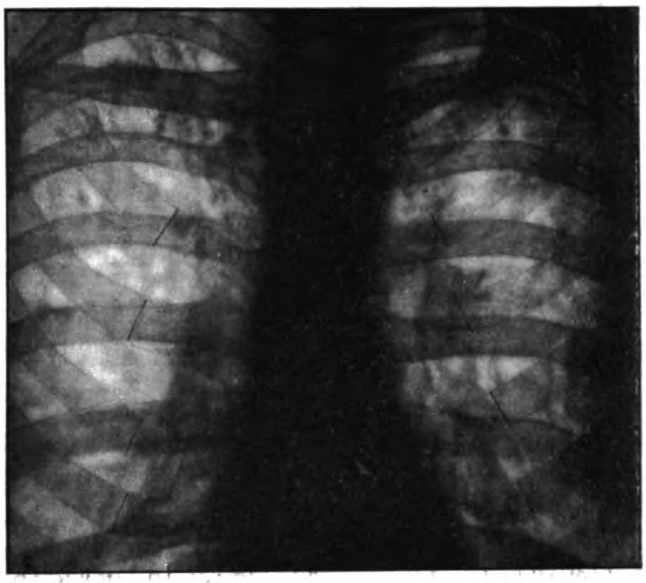


FIG. 9.—Skiagram from case of chronic fibro-caseous tuberculosis (supposed asthma). Case of gentleman, æt. 39 years, with twelve months' history of asthmatic attacks. Had occasional cough and expectoration, which contained tubercle bacilli. The physical signs consisted of a few crepitations at the right anterior apex, numerous rhonchi scattered over both lungs, and impaired expansion of both sides of the chest. Note the elongated cardiac opacity, the extensive hilus blotches on both sides, the well-marked striation and numerous rounded opacities in both lungs, and particularly the finger-like processes extending from the hilus to the right apex. (Radiogram by Dr. Ironside Bruce.)

(4) *Acute miliary tuberculosis.*—Evidence is accumulating in support of C. Weigert's hypothesis that miliary tuberculosis, whether pulmonary or general, is not only due to a blood-infection, but is often associated with tuberculosis of blood-vessels, and probably the most important sites of such vascular tuberculosis are the branches of the pulmonary veins. Caseous bronchial glands not infrequently become adherent to the adjoining pulmonary veins, penetration of which may lead to systemic infection. In rare instances a large caseous glandular or other focus may actually erode the vessel, thus discharging into the blood-stream an enormous quantity, not only of bacilli, but of their toxins also; such a phenomenon would readily explain the occasional extensive

diffusion of the infection, involving in some instances the tissues of the whole body (including the peritoneum and meninges), and would also account for the high pyrexia due to profound toxæmia.

There is also the possibility at any time of a large number of bacilli being discharged into the thoracic duct or right lymphatic duct, and passing by way of the large veins at the root of the neck into the right side of the heart and so into the pulmonary circulation.

Diagnosis.

While the symptoms of quiescent hilus tuberculosis are practically *nil*, those of active hilus tuberculosis are by no means characteristic and are almost identical with those of slight bronchial catarrh, being differentiated only by their persistence and a more marked degree of failure of health. Certain symptoms are suggestive—obstinate dry cough, dyspnœa, slight hæmoptysis, feeling of tightness in the chest, wheezing (? asthmatic) attacks (particularly in children), occasional evening rises of temperature and increased frequency of pulse in conjunction with a varying degree of anorexia, languor, anæmia, debility and loss of weight.

As regards hæmoptysis, it is not uncommon to meet with patients in whom a pulmonary hæmorrhage has been the one and only symptom suggestive of pulmonary tuberculosis. In not a few of these cases, particularly when the patient has been placed under suitable hygienic conditions, no symptoms ever develop and doubts are often expressed as to whether the patient had tuberculosis at all. I believe that many of these cases belong to the category of central tuberculosis starting in the root area, in which a caseating focus has accidentally ulcerated into a small blood-vessel.

As regards physical signs, the disease is so deeply situated that it may reach a fairly advanced stage before giving any external indication of its presence, as it is only the superficial portions of the lung that are really accessible to the ordinary methods of clinical investigation. In this respect hilus tuberculosis resembles central pneumonia, and resembles it still further in the fact that the occurrence of a few crepitations at any one point often indicates, not a small superficial lesion, but an extensive deep-seated one, which at this particular point has reached the surface of the lung. The hearing of a few such crepitations in the infra-clavicular region or supra-spinous fossa is apt to mislead the physician and induce him to diagnose early apical tuberculosis when in reality he may be dealing with advanced central disease (see Fig. 3) with an apical extension. Just as an experienced dentist will diagnose extensive dental caries from the observation of a single black speck situated on the surface of a supposed healthy tooth, so in like manner a careful physician will often suspect extensive hilus and peri-hilus tuberculosis from the presence of apical signs in association with impaired movement—a suspicion which can be readily confirmed or negatived by a modern X-ray examination.

In a certain proportion of cases I have obtained important evidence from a careful examination of *the interscapular region*. It is not uncommon to find close to the root of the spine of the scapula impaired percussion, harsh bronchial breathing, prolonged expiration and increased voice-conduction (bronchophony and pectoriloquy (see Fig. 8). In some instances there is deficient air-entry at one base, while basal rhonchi, for the most part unilateral, are by no means infrequent. My experience leads me to believe that the presence of *persistent rhonchi in a suspiciously tuberculous case often indicates central tuberculosis spreading from the hilus.*

Deaths from bronchitis in middle-aged persons are rightly attributed by insurance offices to pulmonary tuberculosis.

In cases with recent tuberculous deposit there is generally a well-marked tuberculin reaction, which confirms the diagnosis by indicating the presence of some active focus of disease.

In all instances where deep-seated disease is suspected recourse should be had to *radiography*, which *is by far the most important and often the only method of diagnosing hilus and peri-hilus tuberculosis.* Indeed it is practically impossible to form any adequate opinion of the extent of central disease apart from X-ray examination. To my mind, one of the greatest services rendered by radiography consists in the valuable information it affords in regard to morbid thoracic conditions.

It is indeed quite remarkable what advanced broncho-pneumonic and fibroid changes may be revealed by the fluorescent screen and radiogram, quite unsuspected even after a most careful physical examination, in cases which were presumed either to be quite in the incipient stage or free from active disease altogether. This is particularly the case when the condition is complicated by emphysema of the lung. The hyper-resonant percussion-note masks any underlying dulness, while the accompanying bronchitic signs (whether rhonchi or *râles*) make it impossible to differentiate the adventitious sounds characteristic of tuberculous infiltration, although the concentration of bronchitic signs in the upper parts of the lungs may make one suspicious of serious underlying disease. Fortunately these cases, so difficult to diagnose apart from the presence of tubercle bacilli in the sputum, are readily diagnosed by X-ray examination, as the clear emphysematous lung-tissue stands out in marked contrast to the deep mottlings, blotches and shadows of tuberculous disease (see Figs. 9 and 10).

I have already discussed the characteristic X-ray appearances, including hilus blotches and linear shadows indicating fibrous thickening at the root, and the mottlings and clustered opacities of active tuberculous broncho-pneumonia.

Blotches, opacities and linear shadows at the actual root are so common as to be considered almost normal—"the normal hilus shadows." On the other hand, shadows outside the hilus proper are much more significant, and definitely mottled or stippled areas in the peri-hilus tissue, particularly if they assume a clustered or racemose form, are highly suggestive of active tuberculous disease.

I have referred to the difficulty of diagnosing tuberculous infiltration at the roots from silicosis, and other forms of pneumokoniosis, and from non-tuberculous chronic inflammatory deposits.

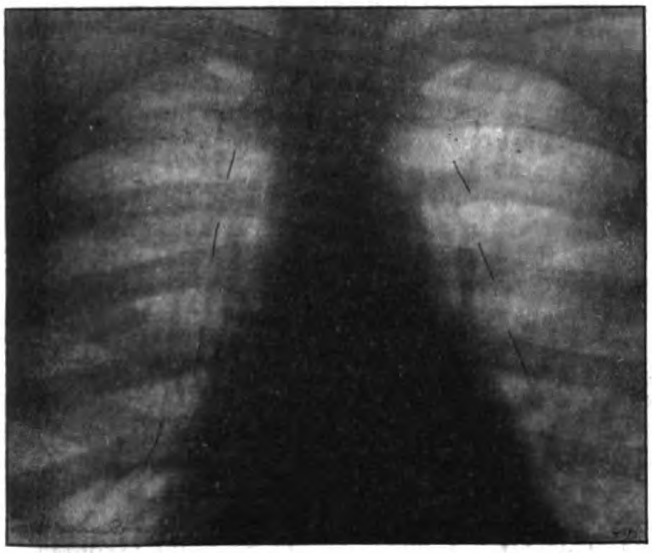


FIG. 10.—Skiagram of case of silicosis, from gentleman, æt. 40 years, who had been exposed for many years to inhalation of silicious dust in South African gold mines. Note the increased opacity of both lungs, more particularly in the region of the hilus, with well-marked linear shadows extending outwards into the emphysematous lung tissue. Was for some time troubled with spasmodic cough. After two years in fresh air all symptoms disappeared, and patient is now quite well. (Skiagram by Dr. Ironside Bruce.)

My experience leads me to confidently state that it should be made a definite rule for all children and young adults who manifest the symptoms of persistent bronchial catarrh, particularly if accompanied by anæmia, lassitude, or other signs of failing health, to be submitted without delay to an X-ray examination.

PROGNOSIS.

The earliest stage of hilus tuberculosis is one of the most curable of all morbid conditions, as is proved by the fact that the great majority of all lungs exhibit post-mortem evidence of healed tuberculosis in the root area, whether in the form of calcareous deposit or fibrous thickening. In the case of hilus tuberculosis not strictly confined to the root area, but extending into the immediately surrounding pulmonary tissue—the peri-hilus—the prognosis is as a rule extremely favourable if the patient's environment is a suitable one. When, however, the disease has reached the apex or other superficial portion of the lung giving rise to physical signs, the outlook is much less hopeful.

Generally speaking a good prognosis depends mainly on two factors:

(*a*) *The degree of resistance of the individual.*

(*b*) *The early diagnosis of the morbid leison.*

(*a*) The degree of resistance depends largely on the comparative immunity and phagocytic efficiency derived from a good heredity, and also to a great extent on favourable environmental conditions. The ubiquitous tubercle bacillus probably obtains lodgment in the pulmonary root area of each and all of us sooner or later, and yet in over 80 per cent. of us the natural resistive power of our tissues is sufficient to effect a permanent cure. This proves the paramount importance of the natural resistive forces of the normal healthy individual. That environment as well as heredity plays an important part is proved conclusively by the fact that tuberculosis claims its victims mainly from the poorly fed, poorly clothed and poorly housed members of the community.

The importance of environment is still further borne out by a comparison between hospital and private patients suffering from tuberculosis. Thus in Dr. Pollock's Brompton Hospital statistics the average duration of life was two and a half years, as compared with Dr. Theodore Williams's statistics taken from the records of a thousand consecutive private patients, whose average duration of life was seven years.

(*b*) As regards the *value of early diagnosis* it is impossible to over-estimate its importance. The late Dr. Gee was of opinion that "if physical signs were present the probabilities were against complete recovery, but if the disease were detected before this there was hope that it might be cured." This statement is as true now as it was twenty years ago. In modern radiography we have a method of diagnosing early cases of pulmonary tuberculosis that Dr. Gee in his day did not possess, it being now possible to discover deep-seated tuberculous disease in the hilus and surrounding pulmonary tissue before tubercle bacilli appear in the sputum and long before physical signs manifest themselves.

Dr. Jordan states that in favourable cases of what he describes as "peri-bronchial phthisis" the X-ray mottling of the lung tissue, indicating active foci of disease, becomes replaced by linear shadows due to the broncho-pneumonic areas cicatrising into fibrous tissue. In progressive cases, on the other hand, the mottling becomes more pronounced

and definite blotches appear due to coalescence of the mottled areas, while in some places the linear shadows may become wider and more clearly defined, due to the accompanying fibrosis. The combination of these two appearances indicates the presence of definite chronic fibroid phthisis, which sooner or later spreads to the apex and superficial portions of the lung. The co-existence of a few crepitations at one apex with well-marked retraction of the affected side of the chest generally indicates advanced fibroid central disease with an apical prolongation—an opinion which may readily be confirmed by an X-ray examination, which will reveal well-marked central blotching and probably "roof-tiling" of the ribs and costal cartilages. A permanent cure sometimes, though rarely, occurs even at this advanced stage, when the whole lung seems to undergo fibrous transformation as in those cases described by the late Sir Andrew Clarke under the term, "non-tuberculous (?) fibroid phthisis." Such cases are usually met with only amongst the better-off classes who are able to secure specially favourable environmental conditions.

Stages of Hilus Tuberculosis.

The stages of pulmonary tuberculosis commencing at the hilus may be classified as follows:

First stage: Pure hilus tuberculosis.—Hilus tuberculosis strictly limited to the tracheo-bronchial glands and the pulmonary tissue of the root area.

Second stage: Peri-hilus tuberculosis.—Hilus tuberculosis spreading some distance outside the root area in the form of linear prolongations, and accompanied by a few scattered foci of tuberculous broncho-pneumonia.

Third stage: Chronic fibroid phthisis.—There is usually extensive central fibrosis and numerous coalescing, and, it may be, caseating patches of broncho-pneumonia, with extensions to the apex or elsewhere, with or without excavation.

The prognosis in the first two stages of hilus tuberculosis is eminently favourable, granted moderate resistive power on the part of the patient and a suitable environment.

In the third stage one can, as a rule, only hope for quiescence and temporary arrest of the disease, though the life of the patient may be greatly prolonged if the disease remains mainly unilateral. The so-called "one-lung cases" belong to this group. Radiography and post-mortem evidence, however, have made it clear that when one lung has reached the stage of advanced chronic fibroid phthisis there are almost invariably hilus changes in the other lung, glandular or otherwise, although the hilus blotches of the apparently sound lung may not in every case be apparent owing to the fact that it may be dragged beyond the middle line and be concealed by the normal median shadow. A consideration of the symptomatic manifestations in conjunction with occasional repeated X-ray examinations will enable the physician to accurately gauge the progress of the disease in these cases.

Treatment.

Little need be said under this head except to emphasise the importance of commencing treatment at the earliest possible stage of the disease. Successful treatment is thus intimately linked up with the question of early diagnosis.

Dr. Gee's aphorism that "therapeutics must begin before physical signs are developed, and if you wait for physical signs you wait too long," exactly sums up the situation.

Professor Karl Pearson's conclusions in respect to the comparative futility of hygienic environmental treatment, as deduced from the after-history of sanatorium patients, are largely fallacious, for the simple reason that fully 90 per cent. of patients admitted to sanatoria are already beyond the curable stage, although many of them are presumed to be in an early stage simply because no definite physical signs can be detected except at one apex, regardless of the probability of extensive underlying central disease.

If a patient suffering from hilus tuberculosis in an early stage is placed under favourable environmental and hygienic conditions, whether at a sanatorium or elsewhere, the active condition as a rule rapidly subsides, and a permanent cure in process of time is effected, so that calcareous nodules and linear cicatrices are left as the innocent witnesses of previous infection.

If, on the other hand, the cases are not taken in hand until physical signs develop, it is generally too late to effect a permanent cure, as an extension to the apex too often means the presence of more or less extensive tuberculous deposit intervening between the root and the surface of the lung.

It is the characteristic X-ray appearances in conjunction with a confirmatory positive tuberculin reaction to which we must trust in deciding when to commence treatment.

It is in these cases of early hilus tuberculosis that carefully administered tuberculin treatment achieves its most signal triumphs. The increasing thereby of the protective properties of the blood and tissues at this stage will serve in most cases to completely check further progress of the disease and pave the way to a permanent cure. This is particularly so in the case of glandular tuberculosis in children. This is the time for strengthening the resistive power of the organism, whether by hygienic methods or by specific tuberculin treatment, or better still by both methods combined.

I would conclude by expressing the belief that the universal recognition by the profession of the importance of hilus disease and its early diagnosis may inaugurate a new era in the treatment of pulmonary tuberculosis.

January 20th, 1913.

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LISTER'S WORK AND TEACHING.*

By SIR W. WATSON CHEYNE, Bart., C.B., F.R.S., F.R.C.S.,

Senior Surgeon and Professor of Clinical Surgery, King's College Hospital.

Gentlemen,—In taking the chair to-night I have, in the first place, to thank you for the honour you have done me in appointing me your President. This honour is especially great when we look at the list of distinguished men who have already occupied this chair and when we remember that this is the oldest medical society in London, and, I presume, in this country. It was established nearly 140 years ago, and still retains its vitality and position as a very important society. It has always held a high position and it is interesting to note that in spite of the existence of other societies, most of the leading men in the profession have been members of the Medical Society. As time goes on the old members disappear, but their vacant places have always been adequately filled by the fresh generation, and the work of the Society goes on as well as ever. It is but seldom that men appear whose places cannot be quickly and satisfactorily filled by others, but during this last session we have lost one such man: I refer, of course, to the late Lord Lister.

I propose to make a portion of Lister's work the subject of the few remarks I shall make to-night, but before doing so I may refer very briefly to some others who have also died during this year, and whose deaths are a great loss to this Society and to the profession in general.

Sir William Allchin became a member of this Society in 1878, and died last February. He was President in 1901, having previously filled the offices of vice-president and orator, and for the last eighteen years he acted as Honorary Librarian

* Presidential Address delivered before the Medical Society of London.

and rendered most valuable services to the Society in that capacity. During the term of his presidency he presented the gold enamelled badge, which, as President, I have now the honour of wearing.

Another very eminent member of this Society died about a year ago in Dr. Hughlings Jackson, who also was an old President of the Society, having filled that office in 1897, after having previously held the offices of vice-president and orator. His work is classical, and so well known that I need not refer to it.

About a year ago we had to regret the loss of Dr. Pavy who was a striking example of a man carrying on his work to the very last, even though at the time of his death he was over eighty years of age. He was Vice-President of this Society, and delivered the Lettsomian Lectures as long ago as 1860 on "Certain Points connected with Diabetes," a subject which absorbed a great deal of his energies, and with regard to which he was actually making further investigations when he was seized with his fatal illness.

Scarcely two months ago we had to deplore the loss of Mr. Clinton Dent, a man regretted by all not only for his scientific attainments, but also for his kindly and social qualities, which endeared him to all with whom he came in contact. He was elected in 1883, and was thus a member for nearly thirty years, and had served on three occasions on the Council of this Society.

Among other names that I may mention are those of Sir Frederick Wallis, who was a distinguished surgeon, and had especially devoted his attention to rectal diseases; he had been honorary secretary of this Society and also a member of Council. Quite recently we lost Mr. Leonard Bidwell, who had acquired a well-merited reputation as a surgeon, especially in abdominal work, and whose early decease is much regretted; he has also been a member of the Council. Another member of the Council who has died during the past year is Dr. J. E. Ranking, who always took a great interest in this Society. Among others who were not so actively engaged in the work of the Society I may especially mention Dr. William Murrell, a physician at Westminster Hospital and a distinguished therapeutist.

During this year, not only this Society, but the medical profession all the world over, have sustained a very great loss in the death of Lord Lister, who was elected an ordinary Fellow in 1878, the year after he came to London, and in 1904 the Society did itself and him the honour of electing him an Honorary Fellow. Lord Lister was by no means a passive member of this Society, but was greatly interested in, and actively contributed to, its work. As you are aware, he left his medical library to this society, a collection which contains many interesting and valuable works. His first contribution was made to this Society in 1883 on the subject of the "Treatment of Fracture of the Patella." This was followed by two communications on the subject of antiseptic dressings, his work at that time being chiefly concentrated on trying to find an efficient and at the same time a non-irritating dressing for wounds. The first paper on this subject was communicated in 1884 on "Corrosive Sublimate as a Surgical Dressing," and the second in 1889 on "A New Antiseptic Dressing," the double cyanide of mercury and zinc dressing which is still used by many surgeons. In 1891 he delivered the oration of this Society on "The Coagulation of Blood in its Practical Aspects." In that oration he gave a short account of the work which he carried on many years before, and of fresh facts which he had since ascertained.

On looking back over the list of addresses made by your Presidents on their election, I find that they have had reference to a great variety of subjects, some connected with the particular work which the President was chiefly interested in, others referring to some general points of medical education or to the history of the Society itself. It seems to me that perhaps I might interest you most if I take up the few minutes which are at my disposal by referring particularly to one or two points with regard to Lord Lister's work. I need not go into his qualities as a man, his wonderful mental acumen, his great humanity, or the great work which he did in revolutionising the practice of surgery. Unlike many great pioneers, his work has been appreciated to a considerable extent while he still lived, and the impressive ceremony at Westminster Abbey on the occasion of his funeral was a striking testimony to the fact that the value of his work has been recognised, not only by his profession and by his country, but by men of all professions and of all countries.

There is, however, one phase of his work which

I do not think is properly appreciated, and which I fear may be less recognised as time goes on, and that is that in addition to all his great qualities and his wonderful scientific attainments, he was one of the most progressive and careful practical surgeons of his day, and has exercised an enormous influence, not merely in regard to the question of asepsis, but also in the advance of actual surgical operative practice; in fact, in the early days he was decades ahead of his fellows in the actual surgical treatment of his patients as apart from the treatment of wounds.

As soon as he had satisfied himself that he was on the right track with regard to the abolition of septic diseases, and that he could reckon with considerable certainty on excluding sepsis from wounds, he realised the enormous revolution that this discovery must make in the actual practice of surgery, and the enormous expansion of surgical work which must follow as the result of its general adoption. He therefore no longer followed the old routine of treatment, but reconsidered the treatment of every case, to see in what way it might be improved, seeing that sepsis need no longer be feared. This part of his work was one which he never would publish. He did, it is true, publish at a later period a few papers on his practical work, such as the one which he communicated to this Society on fracture of the patella, to which I shall presently refer, but he resolutely declined to publish individual cases or the individual advances which he made in practical surgery. I have often urged him to do so, and have offered to collect the material for him if he would only publish it, and failing that, to act as Boswell to his Johnson if he would allow me to publish from time to time records of the actual practice he was carrying out, but his reply was always in the negative. The great objection in his mind was that he felt that till every surgeon was fully convinced of the necessity for asepsis, and of the value of the methods which he advocated, and was efficiently skilled in them, it would be a very serious matter to publish successful results of operations which it would be practically criminal to perform unless complete asepsis were secured. He feared that the publication of such cases might induce others, who were neither convinced of the necessity for the aseptic precautions which he advocated, nor skilled in that work, to perform similar operations with terrible disaster to the patient. He could not but feel that if this occurred he would be a party to such disasters, and that such results would reflect on and prevent the extension of the new method of treating wounds, which he looked on as a matter of much more vital importance than the introduction of new methods of dealing with surgical cases. He also felt that the system which he was trying to perfect was such a very big and essential thing that he did not wish to distract attention from it by minor publications, and he held that the improvements in surgical treatment which necessarily followed on the abolition of sepsis would naturally occur to anyone who was engaged in active surgical work as soon as he had satisfied himself that sepsis in wounds was no longer to be feared.

I remember a very striking example of the difficulty in which he was constantly being placed in giving advice on surgical cases when he himself was not to be the operator. It occurred soon after he came to London, and though you will no doubt consider his action very unprofessional, you will readily realise his point of view. A patient came to see him with bad cancer in the breast and infection of the axillary glands and one or two glands in the neck. I happened to be doing some experiments for him in the little room behind his consulting room, and he asked me to come in and see the case, so that I heard all that was said. He decided in favour of operation, and advised it strongly. The patient then said: "Very well. Mr. ——" (mentioning the name of a leading surgeon who was violently opposed to Lister's methods and was always speaking against them) "generally attends our family, and I shall tell him what you say and ask him to perform the operation." Lister's consternation and distress were great, and evidenced by the flushing of his face, the peculiar movement of the lips and so on, which were so familiar to his intimates as signs of difficulty. He knew that if this surgeon did this operation which he advised, and it was very possible that he would, if only to show that he was not behindhand, it would in all probability result in violent sepsis and a fatal termination, and he felt that his advice, instead of benefiting the patient, would send her to her death. After much hesitation he plucked up courage and said that under those circumstances he would strongly advise her not to be operated on. No doubt such an occurrence at the present time

would be probably rightly construed as an attempt to secure the patient for himself, but I can assure you—and those who knew Lister will have no hesitation in believing me—that no such thought ever entered his head, and that he was simply imbued with a sense of his great responsibility to his patients, and the necessity of giving them his conscientious opinion under the circumstances in which they were placed.

I think another reason why he would not publish his cases was that at the back of his mind there was also the feeling that if he did publish details of his practice, which in every department of surgery was so new and startling, it would have the appearance of glorification of his practical work over that of other surgeons; he was of a very retiring disposition, and self-glorification was not one of his failings. In his paper on "Fracture of the Patella" in your 'Transactions,' he referred to a patient with ununited fracture of the olecranon who had seen eighteen surgeons before consulting him, and with regard to this he makes the following remarks, which illustrate exactly his way of looking at these matters:

"I have referred to a case of ununited fracture of the olecranon where eighteen surgeons had been previously consulted. I trust no one here will suppose that I mentioned this circumstance for the purpose of glorifying myself. I mentioned it in order to emphasise what I believe to be the truth, that by antiseptic means we can do, and are bound to do, operations of the greatest importance for our patients' advantage, which, without strict antiseptic means, the best surgeon would not be justified in recommending. How wise those eighteen surgeons were in counselling against operative interference, provided they were not prepared to operate strictly antiseptically, I think we must be all agreed. As regards the operative procedure in that case, it was of the most simple character; any first year's student could have done the operation exactly as well as myself, and therefore I trust I shall not be misunderstood by its being supposed that I came here to extol my own skill. That which justified me in operating in that case was simply the knowledge that strict antiseptic treatment would convert serious risk into complete safety."

At the same time, although he did not publish his cases, his work was done openly, not only before the students but also before surgeons, who flocked to see his practice from all quarters of the globe. As a consequence his methods of dealing with various surgical conditions became widely known, and many of the advances which were in reality made by Lister are now attributed to other surgeons.

I think the best way in which I can give you some idea of the sort of practice that Lister was carrying on in the early days will be to give you a few extracts from notes which I took of his lectures during the winter session of 1872 and 1873 and the summer session of 1873, that is to say, forty years ago. These notes are very crude, but I may say in excuse for this that at the time I attended the lectures from which I shall quote, I was far too junior to appreciate or understand all the points. I had only completed botany, zoology, and chemistry, and had attended one course of anatomy; I had not yet done any practical surgical or medical work. I was just commencing the elementary courses of surgery and physiology, and I knew nothing whatever about pathology. As a matter of fact I only attended Lister's lectures during that session because the hour at which his lectures took place was one in which I had nothing else to do, and as the time was too short to go to my lodgings and come back, and as the students in those days had no reading or retiring room at Edinburgh University, I simply went to Lister's lectures as a place to sit down till my next lecture was due. I had not, however, listened to the whole of the first lecture before my enthusiasm was awakened, and I felt that I was listening to something which was too good to forget. The habit of students in Edinburgh in my time (and I am distressed to see that it is not the habit in London) was to take copious notes of the lectures, and as Lister spoke very deliberately and slowly, and as I happened to be a rapid note-taker, I was able to take down practically all that he said. Further—and this is an example of the influence which Lister had on his hearers, an influence not confined to myself or only a few of his students—I may say that when I went home in the evenings I sat down and wrote out more fully and more completely what I had heard, so that at the end of the session I possessed a fairly complete record of his lectures written out in a legible manner. These notes are still my most treasured possession, and I am always filled with admiration

and reverence when I look through them and realise how completely he was able to disassociate himself from the teaching of his day, and how sound his views of forty years ago still remain as judged by modern standards.

In this notebook I have a record of 104 cases of various kinds, practically exemplifying all the ordinary departments of surgery with the exception of abdominal surgery. As a matter of fact at that time, except operations for intestinal obstruction and strangulated hernia, there was no abdominal surgery, as we know it, done in the general wards; ovariotomy, which was the main intra-abdominal operation, was performed in special rooms. Although many of these 104 cases are of the greatest interest, I shall only make a selection of four subjects to illustrate the sort of work that was being done.

Suprapubic Lithotomy.

Perhaps the most interesting of all the subjects which he considered during that Session, and that which best exemplifies the way in which he had begun to look at the surgical treatment of those days from a new point of view, is the question of suprapubic lithotomy. I have often heard it asked who it was who re-introduced suprapubic cystotomy. I say "re-introduced," because it was performed many years ago in cases of stone in the bladder, especially by Cheselden, but it had practically entirely disappeared from surgical practice and had been displaced by lateral lithotomy: in fact, forty years ago there is little or no mention of suprapubic lithotomy in books on surgical practice. When, however, the fear of sepsis in ordinary wounds was overcome, a consideration of the two operations led Lister to advocate suprapubic lithotomy in preference to lateral, and the following are the remarks which he made on a case which he discussed in one of his lectures in November, 1872.

The patient was a boy, æt. 14 years, with a stone in his bladder, which gave rise to very severe symptoms, and after Lister had discussed the symptoms of calculus and the various kinds of stones he came to the subject of the treatment. He first referred to lithotrity, but said that in this case this procedure was not advisable on account of the age of the boy and the irritability of the bladder. (This was before the days of litholapaxy.) He then pointed out that there were two operations for removing stone from the bladder, viz. through the perinæum and above the pubis. After describing the steps of the lateral operation he discussed the dangers, mentioning the following—hæmorrhage, shock (especially when large stones have been dragged through the prostate), inflammation in the cellular tissue spreading to the tissues around the neck of the bladder, extravasation of urine and pyæmia (with regard to which he stated that the veins may become the seat of suppurative phlebitis followed by pyæmia). He then made the following remarks on the suprapubic operation, the diction of which I hope you will excuse:

"Now in the hypogastric operation there is no hæmorrhage, no shock, because you do not operate on important parts, not much liability to inflammation, and not much risk of pyæmia, but there is a risk so great that the operation is generally considered unjustifiable, because the cellular tissue being lax and loose the urine is apt to become diffused through it, and cause violent inflammation and diffuse suppuration, which either communicates with the peritoneum or leads to death from profuse suppuration.

"Before Cheselden took to the lateral operation he had performed the high operation nine times, and he had eight recoveries; the ninth patient is said to have died because he washed his hands and face in cold water and also drank cold water; he died of diarrhœa and abscess of the kidney. Another man" (whose name I did not catch) "had ten cases, eight of which were successful; in one the peritoneum was opened, and the other was like Cheselden's fatal case. There are many striking examples of the success of this operation. It seems to have fallen into disfavour because of the advocates of union by first intention. Allanson (?) left an opening for the free escape of discharge during the first twenty-four hours, but his successors sewed up the whole wound. You should, in the hypogastric operation, leave the wound absolutely free and open. Heister had diffuse suppuration, but he used strapping; later he saw that it was wrong to bring the edges of the wound together. In Cheselden's first case he made too small a wound."

"The danger of the high operation is the danger of diffuse suppuration from extravasation of urine. Urine is bland and unirritating unless it is putrid. In a cavity outside the kidney I once found urine

and yet there was no irritation or suppuration. In the child the urine is less irritating than in the adult. In a case of extravasation of urine which I treated antiseptically there was no sloughing. If, then, the high operation is safer, let us see whether it is an advantage.

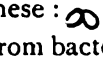
"By means of catgut properly prepared we may stitch the deep parts together and then bring the superficial parts over them and we never see the catgut again. In two cases, after laying open the sac of an irreducible hernia and after having detached adhesions and returned the bowel, I pared the edges of the deep opening, stitched them together with catgut and treated the external wound antiseptically." (This was probably one of the earliest attempts at radical cure of hernia by closing the sac.) "In one of these cases the patient had a fit of insanity and tried the catgut stitches severely, but she has for years had no return of the hernia.

"Now might we not stitch up the opening in the bladder?—that is to say in performing the operation, seize the bladder by artery forceps, cut from above downwards, remove the stone and stitch up the bladder immediately, only stitching together the muscular coat, not the mucous. Now is there any need of tying in a catheter? That there is no danger of forcing urine into the submucous cellular tissue of the bladder has been proved by Simon in vesico-vaginal fistula. After paring the edges and stitching them together, he tied in a catheter, but later he did not do so but had the urine drawn off by a catheter. Sometimes, however, they forgot to draw it off and no harm resulted, and now he leaves the patient to pass water as she pleases. Hence we shall let this boy pass his water, and as long as the urethra is healthy putrefaction never passes back to the bladder."

On November 28th he gave us details of the operation he had performed three days previously. I need not describe these details, except to say that he was not able to distend the bladder satisfactorily and accidentally opened the peritoneum. Whether he stitched up the wound in the peritoneum or not my notes do not say, but he proceeded to extract the stone and then stitched up the hole in the bladder with five catgut stitches not going through the mucous membrane. The boy was allowed to pass water himself. On the morning of the 28th he was able to hold his water for four hours. At the lecture on December 5th we were told that the boy was doing well, but that the stitches in the skin had been taken out, evidently too soon, and afterwards the wound gaped and granulated, but the stitches in the bladder held. The boy left the hospital well on January 6th, 1873.

On May 8th, 1873, he showed us another case of stone in the bladder, and subsequently made an attempt to perform lithotrity, without success on account of the hardness of the stone. He therefore decided to perform lithotomy, and on May 26th he made the following remarks with regard to the suprapubic operation which I may transcribe:

"Now what kind of lithotomy are we to perform? Is this a case for hypogastric or perinæal lithotomy? For the hypogastric operation the urine must be sweet. If we did it with putrid urine the stitches would putrefy and the abscess would discharge itself into the bladder, and during the operation we may let out putrid urine, etc., and have putrid peritonitis, because I believe if we ever establish this operation that it will be safer to cut the peritoneum." (He developed this idea of opening the bladder through the peritoneal cavity a year or two later, but finally abandoned it for the orthodox method. At this time, however, he intended to do it in his next case.) "The mere opening of the peritoneum has, I believe, no material risk. Even without antiseptics it is freely opened in ovariotomy. We would have several advantages by opening the peritoneum. For one thing we would have to stitch the peritoneum, and thus the bladder would be more secure because the peritoneum unites very quickly. Again, by this procedure we get farther from the penis, and we need not provide an escape for discharge because the peritoneum absorbs the discharge.

"In this case, till last night, I intended to perform this operation. But last night I got some of the perfectly fresh urine and it had a peculiar smell, and under the microscope I found bodies like these:  . These are distinct from bacteria in one way—that bacteria divide transversely, while these may divide longitudinally, and these I have called granuligera. Hence we will perform the ordinary lateral operation."

This is very interesting as being probably the first case in which a bacteriological examination of urine was made and in which the result influenced the treatment.

Varicose Veins.

Let me take a second example, namely, the treatment of varicose veins. At that time operation on varicose veins was universally looked on as very dangerous. Various operations had been designed with the view of obliterating the veins without the risk of pyæmia, but they were either unsuccessful or dangerous and deliberate operations on veins were practically tabooed. Now as showing the type of Lister's mind and how he was always trying to imitate the processes of Nature, he saw on looking at the history of varicose veins that spontaneous cure, of individual veins at any rate, often resulted from inflammation of the veins, which was followed by coagulation of the blood and more or less complete obstruction of the lumen of the vein. The first attempt, therefore, which he made in the treatment of varicose veins was to imitate Nature and to try to set up a phlebitis which would lead to obliteration of the lumen of the veins. Accordingly in his lecture on November 7th, 1872, he showed a case of large varicocele in a young man, æt. 26 years, and the following are the remarks he made about the treatment:

"*Treatment.*—A suspensory bandage in general suffices." (Here the palliative treatment had been unavailing). "In addition to this treatment, we may cut away part of the scrotum, and when the wound heals it contracts and acts as a suspensory bandage, but by-and-bye it borrows skin.

"Often if there is inflammation in the veins the blood becomes coagulated and the veins become atrophied, and if the inflammation comes on spontaneously it passes off without material risk and the patient may be cured. Now if we could artificially cause inflammation of the veins we might cause their obliteration.

"Various attempts have been made to excite inflammation artificially, but these have not been satisfactory; one method is to tie the vein. Now any method of exciting inflammation artificially by external treatment is dangerous, because we may not have a harmless inflammation, but inflammation leading to suppuration of blood in the veins—suppurative phlebitis, and as a consequence, pyæmia. One method is to pass needles under the veins and allow them to ulcerate out. Another is to tie the veins with silver wire, and this is often useful and less dangerous than silk. An ear-ring does not cause the irritation that a silk thread would. Threads are put in for the purpose of causing inflammation; these are called setons, and as long as the thread is in the suppuration goes on. Thus, if a rifle ball passes into the tissues it may become surrounded with a capsule of fibrous tissue and there may be no suppuration, but if a part of the clothes be driven in along with the ball we have suppuration. Now the explanation of this is as follows: In the case of the thread the material is porous and becomes soaked with blood and serum, which subsequently undergoes putrefaction, and hence it becomes a source of irritation and causes suppuration. But the wire or bullet, not being porous, affords no nidus for putrefaction, and in the same manner glass remains in the tissues. Hence we see the great influence of putrefaction on wounds, and the results of the antiseptic treatment confirm this, because if a body be introduced among the tissues so as to avoid putrefaction, no suppuration follows. Hence we see how wire is safer than thread. Also from some observations which I" (Prof. Lister) "made some years ago, I found that suppurative phlebitis is due to the introduction of putrid substances into the veins. Every now and then, however, even with wire, pyæmia has been the result, but if we can excite inflammation by antiseptic means it ought to be free from risk, but often the wire does no good."

(*Lecture on November 11th: Varicocele* [*continued*]). —"The tying of the vein also often does no good because the veins become opened up and as large as ever. Obstruction, then, at definite spots is not sufficient. To inject irritating liquid into the veins seems the nearest approach to the natural method of cure, but it is not free from danger. In the earlier stages of the use of carbolic acid, undiluted acid was used and irritated the tissues without irritating too much, and I thought it would not be dangerous to introduce it into the veins. I accordingly tried it in a case of varix of the lower extremity, and extensive consolidation was produced and the patient left in comparative comfort. Three years afterwards there was scarcely any trace of varix and no varicose ulcer. In two other cases no bad effects followed and consolidation took place, but we have not had time to see the ultimate results.

"Now we must prevent the carbolic acid from mixing with the general circulation, because if it is

introduced into the general circulation it would cause general coagulation and death. In the leg I first put on a tourniquet on the limb loosely so as to cause turgescence of the veins without constricting the flow of blood through the arteries, and when the veins are turgid then stop all circulation in the limb, in the arteries as well as in the veins. Then I use carbolic acid, which, if pure, is crystalline, but on adding 10 per cent. of water to it we get liquid carbolic acid. Then by the fine syringe used for the subcutaneous injection of opiates with a fine tubular nozzle some of the liquid is introduced. The piston has a scale on it and also a screw which can be put to a certain point on the scale and thus prevent the piston going further. One half minim is introduced at each place and then a pad of gauze put on to prevent the escape of blood. This is done in several places and an antiseptic dressing is wrapped round the limb. Then I wait ten minutes till all the blood has coagulated and there is no fluid carbolic acid in the veins and then the tourniquet is gradually relaxed."

There are also notes of cases of varix in the leg, treated in the same manner, of which I may mention one. The case (a man) was shown on December 9th and was treated a few days later in the above manner. In the lecture on February 3rd he says:

"In this case the veins so treated are hardly visibly varicose. The coagulum is not yet absorbed, but the limb is not now painful. Here and in a female patient we threw in a whole minim at each place, but in both these cases we had gone rather beyond the desirable limits. There was reaction and inflammation, which in the sailor went on increasing for four days, producing inflammatory fever, but under leeching and warm fomentations the inflammation subsided."

And a further note at the lecture on February 20th says:

"The veins are now hard and occluded, and there is no varix."

In passing I may call attention to the length of time that this patient was in hospital. This was characteristic of Lister. He would not allow a patient to be sent out of hospital till he was quite well, in spite of the protests of the hospital authorities as to the length of their stay. If he undertook the treatment of a case he felt that he was responsible for it to the end. I am afraid few of us are so conscientious.

During the same session he applied this method of treatment to hæmorrhoids and to nævi. In the case of hæmorroids he pinched the base of the pile by forceps, injected half a minim of pure carbolic acid, and some days after clipped off the pile. In the case of the nævus he cut off the circulation by passing a series of threads around it.

It may be in the memory of some that some fifteen or twenty years later the introduction of carbolic acid into hæmorrhoids became for a short time quite the vogue, but here, in 1872, we have Lister carrying out the same practice. I am not aware that those who introduced this method of treatment ever knew or thought that it had been tried and discarded many years before by Lord Lister.

This method of treating varicose veins has quite fallen into disuse, though I still sometimes employ it in nævi. I am not sure that it might not be well to reconsider the plan in the case of varicose veins, for even removal of extensive portions of the veins does not always lead to a perfect result.

Cancer of the Breast.

To take another example, we have the treatment of scirrhus of the breast. At that time the operation for scirrhus of the breast was a very imperfect one, partly because the mode of spread of the disease was not realised, and partly for the reason that while a certain number of patients did recover after the removal of the breast alone through a small incision, anything like an extensive operation was very likely to be fatal; an operation which involved extensive opening up of the axilla was considered to be out of the question. Enlarged glands in the axilla were therefore held to be a contra-indication of operation. On March 3rd, 1873, we have the following notes of a case of scirrhus of the breast which you will see is the foundation of modern practice, and which was far ahead of surgical opinion and practice at the time.

The case was one of scirrhus in a woman, æt. 48 years. The tumour was fairly large, and there were enlarged glands in the axilla. He pointed out the necessity for removing the whole mamma and not merely the affected portion, and also asserted that the enlarged glands are not a bar to

operation, provided that they are all taken away. Having referred to the mode of spread of malignant disease and the secondary infection of the glands, he went on to say:

"Hence we must suppose that the disease or its germs have been carried along the lymphatics and deposited in the glands. Hence the affection of the lymphatic glands in cancer is as much local as the primary disease, because all the parts are affected with the same kind of cancer. Hence if it be right to operate on the primary disease, it is right to operate on the secondary if you can take away all the glands. When the axillary glands, except only one or two, are affected, the case is, however, generally regarded as hopeless."

He then went on to refer to a case of a relative of his own, which he had under his care about four years previously. She had developed a large cancer of the breast with enlarged glands in the axilla. She had been refused operation on account of the axillary condition, and was arranging to go to a quack doctor. Lister went to see her, and persuaded her to wait a few days before seeing the quack. He then went back to Glasgow, and made a dissection of the axillary glands, and came to the conclusion that they could be removed. Relying on his antiseptic arrangements at that time to prevent sepsis, he operated on the patient.

"She lived for two years, and recovered the power of her arm, and ultimately died of cancer in the liver."

Lister's operations for cancer of the breast, though not so complete as they are now, were much more extensive than any others performed at that time, and he made a point of clearing out the axilla pretty thoroughly in almost every case.

Surgery of Bones and Joints.

A good deal of Lister's work at that time was concerned with the surgery of bones and joints, in which again he was far ahead of the other surgeons of his day. He laid the foundation of many operations which are now of frequent occurrence; operations, for example, for deformities, such as knock-knees and deformed tibiæ, for exostosis, for fractures, both recent and ununited, more especially at that time ununited fractures, etc. I have already referred to the first communication which he made to this Society in 1883 on fracture of the patella, and included in that communication were cases both of recent fracture and of old-standing ununited fractures. In that paper he refers to a case of ununited fracture of the olecranon which was apparently the first of this class of cases which he operated on. This case occurred during the session to which my notes refer. The patient came to the hospital at the end of the winter session, and was not therefore shown to the class before operation, but we saw the case afterwards at the lecture on May 15th, 1873, seven weeks after the operation, and by that time the elbow was freely movable. In this case he had not buried the wire, but had left the twisted ends projecting in the middle of the wound. Hence on May 15th the wire was removed, and the patient left the infirmary with his elbow freely movable, and as useful as the other. Later on Lister gave up the plan of leaving the wire projecting, and cut it off and closed the wound.

During this session he also operated on an ununited fracture of the radius and on one of the femur. The latter was a very bad case of fracture just above the knee-joint, with the lower fragment turned back and the patella adherent to the lower end of the upper fragment. I need not go into the details of the operation, which do not materially differ from those of similar operations at the present time. He naturally opened the knee-joint. With the aid of pulleys he gained about an inch in length, and the result was quite satisfactory.

In operating on this case, he mentioned one of ununited fracture of the neck of the femur which he had treated some years previously. Apart from the fact that this was probably the first case of ununited fracture of the neck of the femur which was ever operated on, there is one point of interest in this case, viz. that he pared both surfaces of the bones with a gouge and did not take away any of the chips of bone, because he thought that they would be absorbed. I need not point out that at the present time we sometimes use these chips to fill up intervals between bones.

"Except one spicule of bone, I never saw anything of the chips, and now he is walking on two sound legs."

Coming lastly to the cases of fracture of the patella which he brought before this Society, I need not go into details, but, to show the current surgical opinion in 1877, I may say that one very eminent and leading surgeon, having heard of the

first case on which Lister had operated, remarked to his students, in speaking about the risks to which this new surgery was subjecting patients, that "when this poor fellow dies it is proper that someone should proceed against that man for malpractice." The case was quite successful, and was shown in 1883 at the Medical Society.

I have already taken up more time than I had intended. I might have referred to other very interesting cases which occurred during that session, and even then I would have described nothing like all the work which he was doing, because he only brought before the class cases which he could demonstrate from a distance, and cases which were suitable for men commencing surgical training. When, as I say, you reflect that this was only the new work of one session, and that steady alteration in surgical methods had been going on and continued to go on for many years before other surgeons saw their way to follow Lister's lead, you will realise that his work was not simply limited to the application of bacteriology to surgery, but that as a practical surgeon he was far in advance of his time. There is no doubt that the patients who were fortunate enough to come under his care in his wards had chances of recovery from their ailments such as they could not at that time obtain elsewhere.

As a surgeon Lister was a slow operator, but very careful and thorough, and when the operation was completed one could feel sure that nothing had been neglected to make it a success. He could, however, be quick when necessary, and his amputations were performed beautifully and expeditiously. The department of operative surgery in which I always admired him most, and in which I have never seen his equal, was, however, in plastic surgery. To see him close a defect in the face or elsewhere was a great treat.

Gentlemen, I am afraid I have wearied you, but one cannot be associated so long and so intimately as I have been with such a man as Lister without becoming more or less of a hero-worshipper, and there is so much that can and ought to be said about his work that it is difficult to know where to stop. I said at the beginning that the place of most men is rapidly and satisfactorily filled, but it requires such an extraordinary combination of qualities and abilities to make a Lister that such a man only arises at long intervals. We can only take him as a type and example to be followed, and hope that by following humbly in his footsteps we may add some little thing to the progress of knowledge and for the benefit of humanity.

January 27th, 1913.

THE TREATMENT OF FRACTURES ABOUT THE ELBOW-JOINT.

By GRAHAM SIMPSON, F.R.C.S.Eng.,
Surgeon, Sheffield Royal Hospital.

I MUST start by confessing that my title is misleading; what I really have in my mind is the treatment of fractures of the lower end of the humerus by acute flexion.

For some years I have taken a great interest in this method of treating fractures about the elbow- and knee-joints; I believe in simplicity in surgical methods, and this particular method is simplicity itself.

À propos of this, I would draw your attention to the fact that some of the most striking of the recent advances in the technique of surgery have been in this direction, viz. of simplifying complicated methods.

Perhaps the most noteworthy as well as the most important is the use of iodine in the preparation of the skin for operations; I expect that most of those here have had at some time or other to prepare large areas of skin for operation, and will agree with me that the older method was a time-wasting and tedious ritual. Moreover, the asepsis of the skin depended on the conscience of the dresser or nurse who prepared the patient, and there was nothing to show whether this preparation had been thorough or only perfunctory.

Another good example that occurs to me is the matter of dressings. In my student days, the dressing of a case of hernia was an affair of some pomp and ceremony: besides numerous layers of gauze and wool, a sheet of macintosh was placed to prevent the contamination of the urine, and the surgical mind delighted in the confection of complicated bandages to keep the dressing in place. For more than a year, I have used no dressings in my hernia operations; at the end of the operation, the wound is painted with iodine, and this is again done on the third day. The wounds heal extremely well and always look dry, and the patients suffer no discomfort from the contact of the night-shirt. A certain amount of time and labour is saved in this way in the operating theatre, but the advantage is still more obvious when going round the wards. Lastly, there is the reduction in the

hospital dressing account to be considered. I believe that, in the near future, dressings will be much less used.

I will first read the notes, and show the skiagram of a typical case, and let you see the result for yourselves, and afterwards make some remarks on this method of treatment: I hope that you will accept my statement that this is a typical case, and not one specially selected because it is a good result; I could produce about half a dozen more with as good results, and I do not know of any fractures treated in this way that have not done well.

Mary H—, æt. 5 years, was climbing in October of last year, when she slipped and fell on her right elbow; she was seen by a medical man, who put up the arm in an internal angular splint; she was brought to see me on the following day.

The right elbow-joint was swollen and painful, and there was a good deal of swelling of the parts around; there was a marked prominence on the anterior surface of the arm just above the crease of the elbow-joint, and a deformity like that of a backward dislocation of that joint. A skiagram was taken by Dr. W. H. Nutt, and this showed a backward displacement of the lower end of the humerus (Fig. 1). On the next day chloroform was given, and the arm was fixed in acute flexion by a figure of eight flannel bandage, and the flexed elbow bandaged on to the chest wall. Some difficulty was found in obtaining full flexion, but after repeated attempts the joint gradually yielded. After this had been done the radial pulse on that side could not be felt, the hand looked blue, and there were other signs of defective circulation.

I felt a little anxious about this, and therefore saw the child again an hour and a half later, when I was relieved to find the hand less blue and the circulation improving.

Ten days later I took off the bandage and let

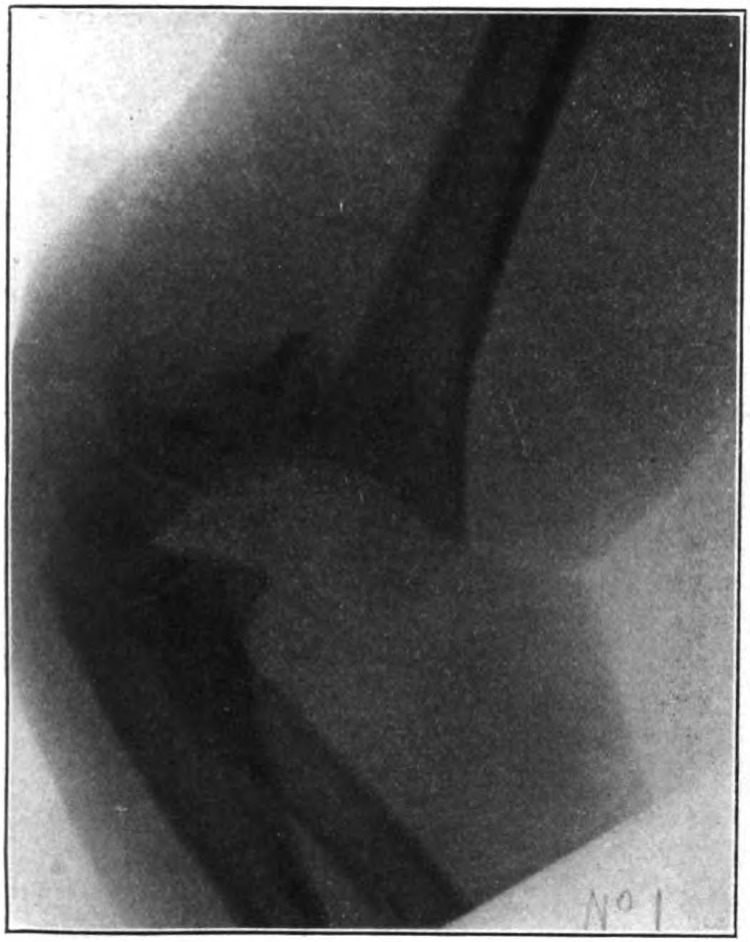


FIG. 1.—Fracture of lower end of humerus before reduction.

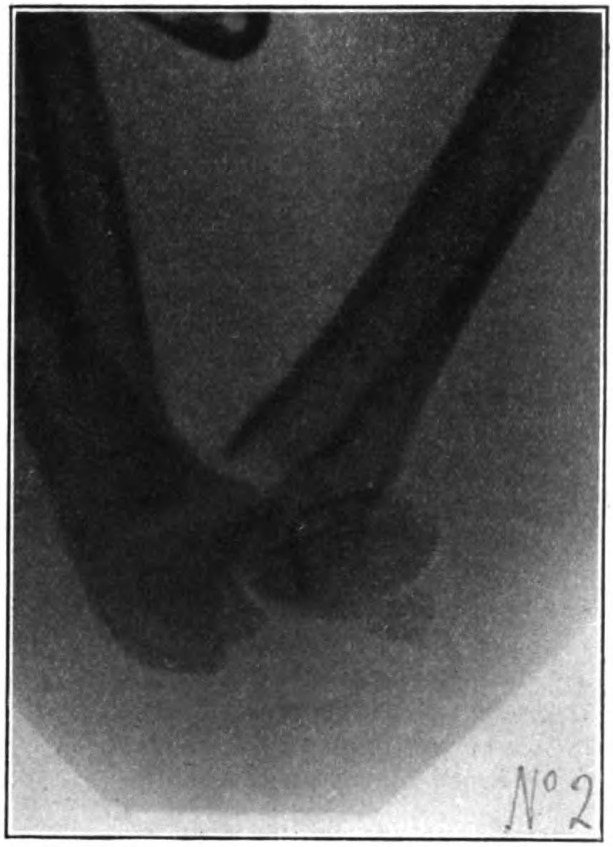


FIG. 2.—Fracture of lower end of humerus after reduction.

the arm come down to a right angle without causing any pain; there was less swelling, but the bruising had extended up the course of the biceps; no passive movements were used, but the child could pronate and supinate the forearm, and had already some flexion and extension of the elbow-joint.

A skiagram was taken, which showed that the position of the fracture was much better, but was not perfect (Fig. 2). I ventured to prophesy, however, that the child would have a good movable joint.

I saw the patient again on several occasions, and each time altered the angle of the joint. Three weeks after the accident the bandage was left off, and the patient encouraged to move the arm. The only other note to add is that she had a course of massage from a professional masseur after the movement had returned.

You will see that she can fully extend that elbow-joint; in fact she can over-extend it, but on examining the other arm you will find that this is the normal condition; she can flex the arm to an acute angle, but not quite so fully as the other arm; for reasons that I will mention later, I think that this limitation will disappear. Her father says that the arm is satisfactory: "she can do anything with it" (Fig. 3).

You will have noticed that the plan I adopt is to have a skiagram taken, then give an anæsthetic, and reduce the fracture; bandage the arm up in acute flexion, and then have another X ray taken to see that the reduction is satisfactory. After ten days I take off the bandage, and let the arm come down to a right angle, and fix it in that position. I then look at the elbow every other day, and put it up each time at a different angle (lately I have omitted to do this, as I think that it is unnecessary). Finally, on the twentieth day, I leave off the bandage and let the child do what it likes with the arm.

This method is not, of course, a new one; in

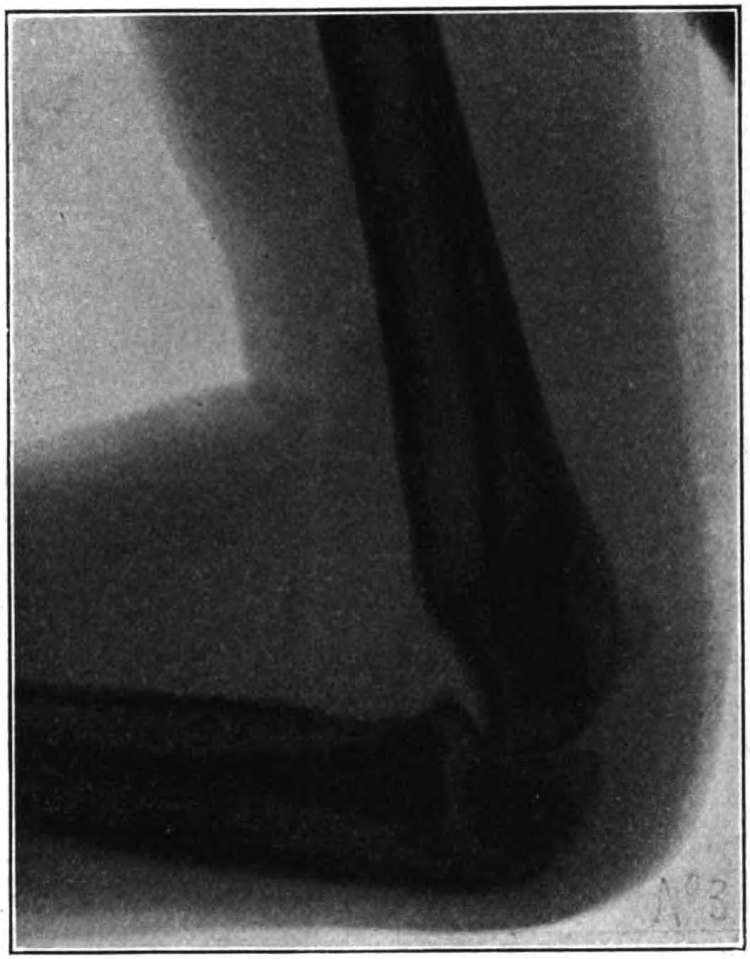


FIG. 3.—Result seven months after accident.

fact, it has nearly attained its majority; yet, though it is well known to every surgeon, it seems to me never to have quite "caught on" with general practitioners; perhaps they fear that the relations will not be satisfied without a display of splints.

I want to draw your attention to the following points:

Its simplicity.—All that is needed is a piece of lint, some chloroform (or, better still, some ether), a mouth-gag and tongue-forceps, some cotton-wool, a bandage, and some common sense.

The importance of giving an anæsthetic.—I think that this is *the* essential point in the treatment; I feel sure that I could not have reduced this particular fracture without an anæsthetic, partly because I should have been influenced by the child's suffering and partly because I should have been working against muscles in a state of spasm.

As a matter of fact, even with complete relaxation of the muscles and with plenty of time, I found considerable difficulty in effecting reduction. There is still another reason: without anæsthesia you will hurt the child so much that you will never regain its confidence.

Passive movements.—You will have noticed the absence of any passive movement in this case. Without going into the question of the process of repair in these fractures, I ask you to accept the statement that passive movements in these cases are cruel and not only useless but they have been proved actually to delay recovery of movement. A remarbable example of this has been recorded by a French surgeon; a child with a fracture of the lower end of the humerus was being vigorously treated by passive movement and the movement of the elbow-joint, instead of improving, rather grew worse; there supervened some medical complaint during which the treatment had to be discontinued and the elbow-joint quickly regained its movement.

Statistics also show that the cases treated without passive movement get moveable joints more quickly than those treated with it.

This statement was very well illustrated by the case of a little girl whom I saw at my out-patients yesterday. Five weeks before she had broken the external condyle of one humerus. The fracture had been conscientiously treated at another institution by means of a splint, which had been taken off every day in order that the arm might be rubbed and the elbow-joint moved. The mother stated that the child had screamed a great deal when this was done. The result was that the fracture had united, but the elbow-joint was practically stiff and in a rather bad position—that of nearly complete extension. Moreover, the child was so frightened that there was some difficulty in even examining the arm. In this case I advised letting the arm severely alone, and I believe that in a short time the patient will begin to try and use the arm, and movement will return.

Massage.—This is actually harmful in the early stages, and should only be used after movement has returned; even Lucas-Championnière, an enthusiast on this question, does not recommend it for these cases.

The final result from the point of view of function.—This has been thoroughly worked out in a large number of cases by several observers; they agree that, even when the result has been unsatisfactory at the end of treatment (*e.g.* two months after the accident), yet if the case is seen some time later (*e.g.* two years after the accident), the functional results as regards movement and strength of the elbow are surprisingly good. This applies to various methods of treatment, but holds good only in the case of children, and depends no doubt on their power of adaptive repair.

The final result from an anatomical point of view.—This is not so good as the functional result. in this case a ridge of bone can be felt just above the crease of the elbow, and is explained by the fact that the second skiagram showed imperfect reduction; this ridge is sometimes present for years. The carrying angle appears in my case to be about normal, but is often found either diminished or exaggerated; its importance from the point of view of function seems to be minimal.

Interference with the circulation.—This is always liable to occur if you flex a swollen arm, but I have heard of only one case in which an untoward result followed: a case of ischæmic paralysis. It was this consideration that made me add common sense to the surgical outfit.

Late improvement,—In the arm I have shown you to-night there is some limitation of flexion; I think this will gradually disappear; it is probably due to the bony spur on the anterior surface of the humerus; as the bone increases in length, this

projection will move further away from the elbow-joint, and the power of full flexion will return.

Skiagraphy.—I am very much in favour of using the X rays in all cases of fracture; not only for finding out the exact nature of the bone lesion, but also to see if the treatment is satisfactory, and to form a record of the case. There is another good reason for taking a skiagram: the omission to do so may be looked on in a court of justice as negligent. I always advise doctors to recommend an X ray in every case of fracture.

At the hospital, I am in the habit of giving the anæsthetic and putting up these fractures in the X-ray room.

At the same time, I feel sure that a skiagram is not essential, and that if the elbow after reduction looks and feels all right, I should not be unduly worried though a later skiagram showed an incomplete anatomical restoration.

Operation.—In these small children, at any rate, I think that operation is not necessary, but I own to having yielded on more than one occasion to the temptation of screwing a fractured external condyle on to the shaft; the operation is so simple and the result so speedy and satisfactory. The treatment by flexion, however, can be adopted under any circumstances, whereas an operation might be very inconvenient. I have not had much experience of these injuries in the adult, but I believe that flexion does not give such good result in their case and that operation is more often indicated.

In conclusion, I wish to emphasise that my object in reading this paper has been to point out to the medical man in general practice how simple this method of treatment is. It is not so much a question of getting a good result as of saving bother to the doctor and unnecessary suffering to the child. There is no necessity for splints or apparatus, nor for those distressing scenes of the doctor trying to move the elbow of a screaming, struggling child with a distracted mother pretending to assist and really hindering him. No difficulty in remembering an elaborate after-treatment; place the arm at a right angle on the tenth day and leave off the bandage on the twentieth day.

I would apologise for reading this paper if it were not that many practitioners, though they must know of this method, are so backward in adopting it; I urge them very strongly to give it a trial.

(1) This paper was read before the Sheffield Medico-Chirurgical Society on April 11th, 1912.

(2) This child now has complete flexion but not quite complete extension.

January 27th, 1913.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from p.* 224.)

(141)

The Ætiology of the Mother-in-law,
or
Altruism and the Menopause.

I take down from my shelves a text-book on therapeutics with regard to which I can gratefully testify that it has afforded me invaluable assistance on many occasions, and I turn with eagerness to the section headed "Menopause." Here I find the whole matter comprised in the recommendation of four drugs, namely, bromide of potassium, cannabis indica, valerianate of ammonium, and as a local application to the head, eau de Cologne. Whereupon I close the book with a sigh and bethink me of the saying of the French physician, to the effect that our therapeutic ignorance with regard to the menopause is undoubtedly responsible for the universal unpopularity of the mother-in-law. In setting ourselves to the study of this question, then, we Æsculapians are undertaking a task, the proper fulfilment of which will cause future generations to rise up and call us blessed, a programme of social reform which is certain to receive the enthusiastic support of both political parties, and one which even the militant suffragette will not venture to relegate to a position of secondary importance.

("The Cardiopathies of the Menopause," by Leonard Williams, M.D., M.R.C.P., 'Clin. Journ.,' vol. xxxiii, p. 325.)

(142)

Pyogenic Affections of the Skin in Children,
or
The Vicarious Philanthropist again.

Talking of pyogenic affections of the skin, Dr. James Galloway says: "In the case of children of school age these diseases are sufficiently common to cause public authorities to recognise that they are deserving of special treatment at the expense of the community, or, as it frequently turns out in such cases, at the expense of the medical profession.

("Common Affections of the Skin: their Exact Diagnosis and Treatment," by James Galloway, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxiv, p. 49.)

(143)

The Value of some of the Modern Methods in Use for the Diagnosis of Pulmonary Tuberculosis.

In the same way I can affirm, from a rather amusing and sometimes very bitter experience during the last five years, abnormal opsonic indices cannot be said to be reliable signs of pulmonary or any other forms of tuberculosis. As for the alleged value of the Von Pirquet and ophthalmo reactions, though very suggestive of the presence of a tuberculous focus somewhere in the body, they are of no serviceable value in attaching a tuberculous origin to the signs discoverable in any organ at the bedside, for the simple reason that these tests may be revealing the presence of a lesion tuberculous in character but placed in some different part of the body to the one yielding physical signs.

("Pulmonary Tuberculosis and Notification," by H. Batty Shaw, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxix, p. 353.)

(144)

Importance of Examining the Sputum.

It is your duty in *every* case in which cough is accompanied by the expectoration of sputum to examine the latter for tubercle bacilli. No doubt it seems an extreme position to take up, but if the experience of others is to be of use to you, you must listen to this piece of advice, and feel that it is an obligation on your part greater and more important than any other examination you would carry out on such a patient. As you will find out, neglect to carry out such an examination will be resented by your future patients, and you will suffer accordingly: you will deserve it.

(*Ibid.*)

(145)

Anomalous Symptoms in a Nerve Case should always suggest Syphilis.

I want you to remember this: that these syphilitic cases should always be suspected when you have patients with a group of symptoms which do not fit in with the regular systematic diseases. I have no doubt you know by heart the symptoms which are peculiar to disseminated sclerosis, to progressive muscular atrophy, to amyotrophic lateral sclerosis, etc. When you have an abnormal case, one which has symptoms which do not fit in with these diseases, think at once of syphilis and inquire about it.

But what is characteristic of syphilis is the indefiniteness and peculiar admixture of symptoms, unlike those of any systematic disease, and especially the dissociation of anæsthesia, and the frequent affections of the bladder without the symptoms of a true transverse myelitis, which wou d account for it by the cutting off of the cerebral influence.

("Syphilitic Disease of the Spinal Cord," by W. P. Herringham, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxix, p. 359.)

(146)

Cacoethes Operandi.

After warmly acknowledging the great indebtedness not only of ourselves, but of thousands of our patients to the surgeon, Dr. David Drummond proceeds: "It seems churlish after this to say anything that may be construed into a complaint, but one almost deplores the fact that operations can be performed so easily and so safely, for seldom do these considerations enter into the question.

Yet I feel we are justified in urging a little more caution, a little more attention to symptoms and signs in the hope of being able shortly to advance beyond the present anomalous position which is a reproach to the profession, viz. that we only too often invite the surgeon to operate in order to decide if the case be suitable for surgical interference. Thoughtful surgeons are raising the question themselves, and I feel that it will not be long before we are subjected to the adverse criticism of the public. Quite recently I was forced to listen to a tirade delivered against the profession by an angry lady, the mother of a patient the subject of consultation, who wound up with—"Oh, the doctors have gone mad over operations," which was all the more trying as I was at the time contending with her for a hearing and attempting to deliver myself of the opinion that her son's case was not a suitable one for operation.

("Is it a case for operation? A Problem in

Diagnosis," by David Drummond, M.D., D.C.L., 'Clin. Journ.,' vol. xxxix, p. 369.)

(147)

A good Surgeon is something more than a mere skilful Mechanic.

"There is no more difficult problem in surgery than that which presents itself in making a proper selection of cases for operation: Anatomical, physiological and pathological knowledge, diagnostic acumen, prognostic capacity, are all requisites of the good surgeon. *Anyone can become a surgical mechanic.* Capable surgeons are not so easily produced. It is so easy and so safe to cut everywhere "to see," that the surgeon is encouraged to operate in any case and in every case. The public applauds because something has been done. The surgeon and the public are apt to forget, until they receive an unpleasant reminder, that it may sometimes be wiser to do nothing.

(Prof. Rutherford Morison: quoted by David Drummond, M.D., D.C.L.; *ibid.*)

(148)

Time of Election for the Operative Treatment of Fractures.

Operation can only be undertaken to the best advantage during the fortnight subsequent to the accident, and during this interval it is all important for the practitioner in charge to convince himself that the position obtained by non-operative means will ensure a satisfactory result, both functional and anatomical, or at once to hand over the patient to a surgeon well used to such operations. I do not see that they can, or ever will be, operations called for from the general practitioner; especially if the consensus of surgical opinion finally condemns immediate operation in compound fractures. If the decision to operate is only arrived at when callus is forming and union is taking place, the most skilful surgeons enter the combat under a most serious handicap.

("The Selection for Early Operation of Fractures of the Long Bones," by C. H. Fagge, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxix, p. 385.)

January 27th, 1913.

Transfusion of Blood.—Henrot remarks that the accidents liable from anaphylaxis on reinjection of serum are not to be feared in injection of the whole blood, and he urges his French *confrères* not to let the Americans re-implant transfusion in France when French medicine already has a fine record in this line. The first of these six cases of transfusion of blood dates from 1876; it was done for uterine hæmorrhage following retention of the placenta in a peculiarly malformed uterus. The patient was almost exsanguinated when 200 c.c. of blood drawn from a man with prominent veins was injected and the woman rapidly recovered. Henrot's experience with this and other obstetric cases indicates that the hæmorrhage from abnormal implantation of the placenta on the cervix, with repeated losses of blood, is a more urgent indication for transfusion than the hæmorrhage which accompanies directly the delivery. It seems as if the organism is able to make up at once for a sudden extensive loss of blood, but that when the losses occur repeatedly the time comes when the blood-producing organs become exhausted and transfusion here is a life-saving measure. In his second case a girl under fourteen with extreme chlorosis and uncontrollable uterine hæmorrhage plus uncontrollable vomiting was saved by transfusion of 170 grm. of blood drawn from her father. There was no further hæmorrhage. In a third case a man, æt. 41 years, intensely debilitated from malaria and dropsy, was injected with 30 c.c. of blood from another man. The number of red corpuscles increased at once by 600,000, and by the end of six weeks the number was 1,890,000, while it had been only 1,082,000 before the transfusion. Seven weeks after the operation all signs of œdema had disappeared and the previously bed-ridden patient was out of doors all the time, but the enlargement of the spleen still persisted, indicating that the patient was only improved, not cured. Faulty technique was responsible for the failure of the measure in the fourth case, the blood evidently having failed to enter the vein as supposed. The veins in these exsanguinated patients are so flabby and collapsed that it is a difficult matter to find and enter one properly. Transient benefit was obtained from the transfusion in a case of pernicious anæmia but the progress of the disease was only temporarily arrested. In another case of pernicious anæmia the patient was a tailor, æt. 30 years, and his blood was like water while the blood transfused was unusually concentrated. The donor was a robust girl cousin from the country, and the blood did not blend. Fatal pulmonary embolism followed in a few minutes. Henrot emphasises that even a small amount of blood may do wonders. He never attempted to transfuse the blood directly but drew it into a Mathieu apparatus for the purpose, a funnel with graduated recipient and rubber-bulb attachment.—*Journ. A.M.A.*, vol. lx, p. 89.

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A CASE OF ANÆMIA OF THE PERNICIOUS TYPE WITH ACHOLURIC JAUNDICE.*

By F. J. POYNTON, M.D., F.R.C.P., and H. C. D. PEDLER, M.R.C.S., L.R.C.P.

WE feel that perhaps we may need to make some apology to the Fellows of the Society this evening for bringing to their notice a case which some may consider one of obvious pernicious or Addisonian anæmia, a disease with which we are well aware they are thoroughly conversant. Yet there is no doubt that it is a case of much interest, and it will serve the useful purpose of ascertaining some of the views that the Fellows hold at the present time upon the exact nature and diagnostic peculiarities of this important disease. In order to bring into prominence some of the points, we shall make some comparisons to the conditions of congenital and acquired acholuric jaundice, diseases which in this country and on the continent have stimulated investigators to discover interesting facts bearing on the obscure problems of blood diseases.

The patient, a married woman, æt. 40 years, was first admitted to University College Hospital, under Dr. S. Martin, in March of this year, and it is to him that we must express our indebtedness for permission to record her case. Except for scarlet fever twenty years ago, followed by an occasional ear discharge, and an operation for ventro-fixation of the uterus four years ago, there had been no serious illness until February, 1910, when she had a severe attack of what was called "jaundice," with fever, prostration and delirium, and for which she was treated with olive oil. From this she recovered, and according to her own account, perfectly. In 1911 she again broke down in much the same way, but the illness was less severe, and on this occasion she was treated with anti-strepto-

* Read at the Medical Society of London.

coccic serum. Once more she recovered, until her third attack, which commenced ten days before her admission to the hospital with vomiting after food of a bilious, frothy fluid which was not blood-stained. The onset was sudden, and the next day "jaundice" was noticed. There was some pain

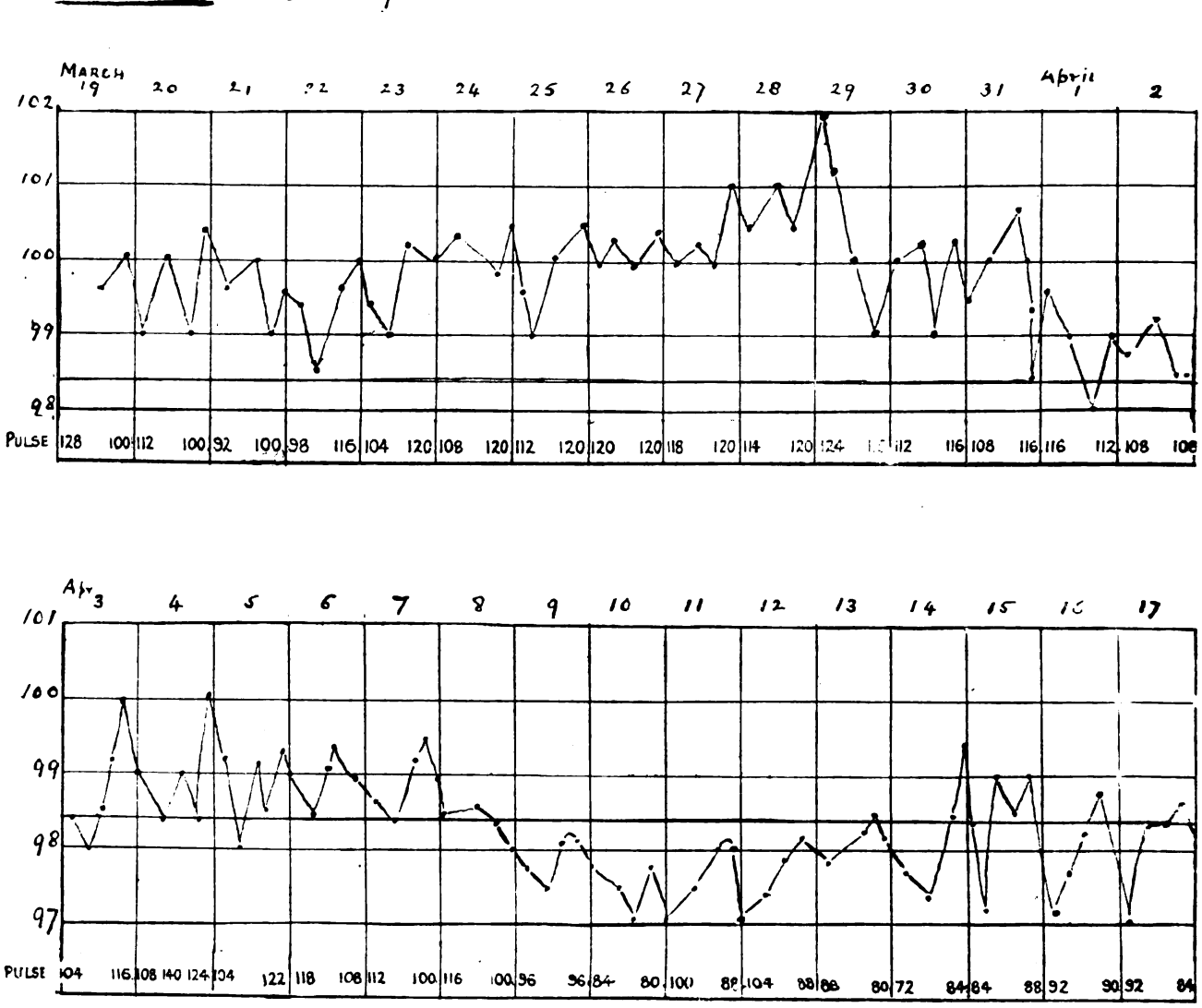

FIG. 1.—The temperature chart during the first illness.

ill on admission, and was so weak as to be unable to walk without assistance. Her skin was a dark lemon-yellow colour and the cheeks had a slight flush. The conjunctivæ showed yellow-green patches at each canthus. The mucous membranes were very pale but neither cyanosed nor pigmented.

with the vomiting, but it was not severe enough to suggest biliary colic. There was loss of appetite and the bowels were constipated. The tentative diagnosis on this occasion had been *malignant endocarditis.*

One additional point may be added to the history, which is that since a miscarriage in the early years of married life she had noticed increasing debility after extra exertion.

A well-developed woman, she looked extremely ill on admission, and was so weak as to be unable to walk without assistance. Her skin was a dark lemon-yellow colour and the cheeks had a slight flush. The conjunctivæ showed yellow-green patches at each canthus. The mucous membranes were very pale but neither cyanosed nor pigmented. The finger-nails were a little blue and the finger-tips shiny and slightly clubbed.

The cardio-vascular system showed the signs of a profound anæmia. The pulse was 104 and irregular in force. Blood-pressure 160 mm. Hg. (Riva-Rocci), falling to 110 mm. There was some cardiac dilatation with apical and basal systolic murmurs. The condition, we thought, corresponded with a profound anæmia rather than an endocarditis.

The tongue was clean and not sore, the teeth,

mostly false, had been well kept. There was no wasting. The liver was slightly enlarged downward, and could be readily felt below the costal margin. The spleen was also readily palpable on inspiration. Except for the scar of the abdominal operation nothing else abnormal was found. On admission the urine was darker than normal, turbid and acid in reaction, specific gravity 1015. No albumen, sugar, or bile-salts were present. The colour was due to uro-erythrin. No leucin, tyrosine or urobilin was demonstrated. The pupils reacted normally to light and accommodation.

The tendon reflexes were not obtained at first, either in the arms or legs. Subsequently both knee-jerks were demonstrated, but always with difficulty. Muscular power was feeble but there was nothing to suggest any paralysis, and sensation to touch, pain, and temperature was normal.

The retinæ were pale, but there were not any hæmorrhages and there was no optic neuritis.

During the first ten days after admission her condition became steadily worse. The jaundice did not change, but her cerebration became slow and her speech deliberate. All night from the first she wandered in mind and then became quite delirious and finally very restless—trying to get out of bed.

The pulse varied from 98–120 to the minute, and at times became very irregular. The temperature, always above 99° F., rose gradually to 102°F. The course of the fever is shown by the chart (Figs. 1 and 2).

The details of the various blood-counts, made by Dr. Embleton, are shown in the tables (Fig. 3).

It is sufficient to note that the occurrence of myelocytes in the first count and the high leucocyte count in the first three suggested a leukanæmia. Also the clear fact stood out that there was an extreme anæmia with a high colour index.

Each day she vomited a little bilious fluid and this followed eating and drinking so rapidly as to be almost a regurgitation of food. Her condition at its worst suggested the last stage of some virulent anæmia and hopes of recovery were of the gloomiest.

During the period of delirium, however, her colour was noticed to be slightly improving, and about the eleventh day after admission she certainly began to mend, and ten days later her temperature had fallen to normal. The vomiting ceased, but

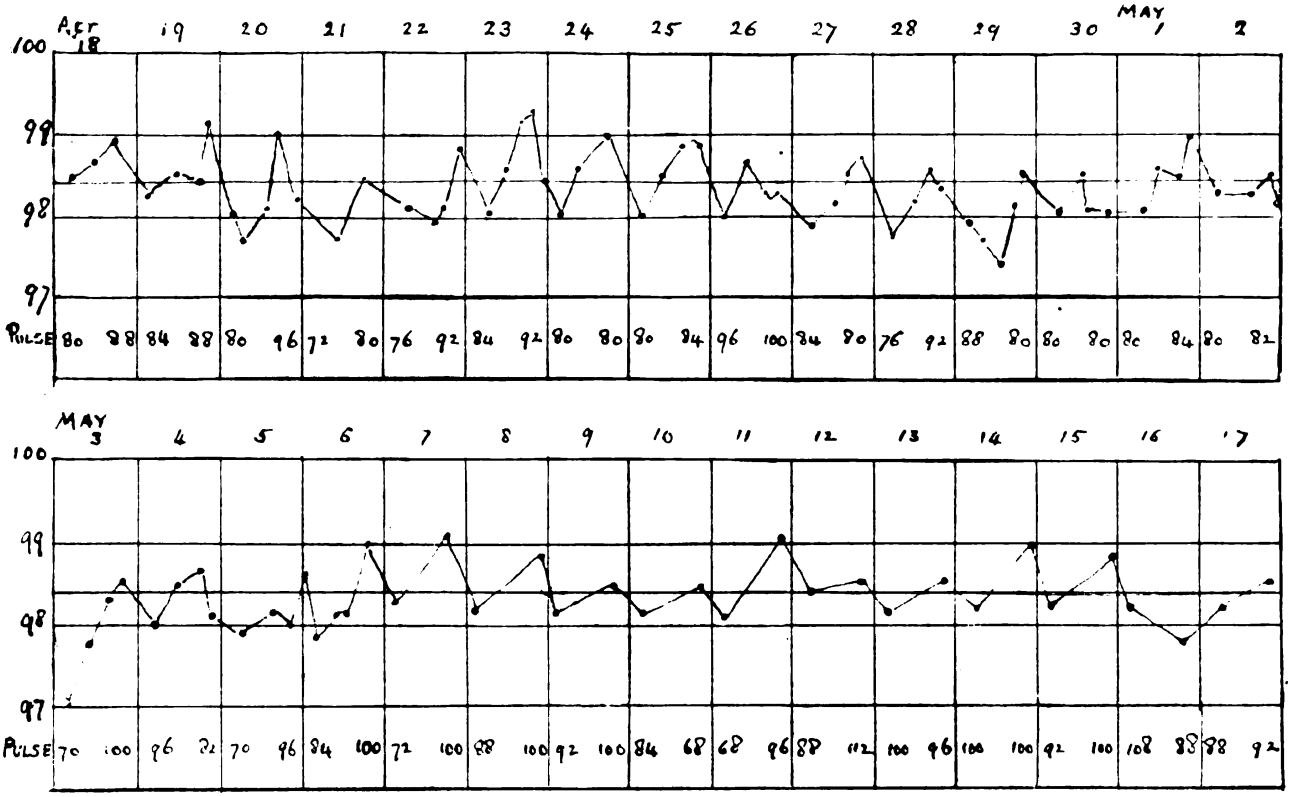

FIG. 2.—The temperature chart during the first illness (*continued*).

the pulse remained still feeble and irregular. Gradually the lemon colour disappeared and the face became highly coloured, even flushed. Delirium and restlessness ceased, and her speech improved. A week later the pulse had dropped to 72–96 to the minute.

Blood-count.—(Fig. 3, No. 2.)

The liver became no longer palpable, but the spleen was more easily felt below the costal arch. The bowels were constipated and menstruation was in abeyance until May. The functional cardiac murmurs became less audible.

Her treatment consisted in rest and appropriate dieting. Arsenic was administered in gradually increasing doses, commencing with ♏j of the liquor arsenicalis. This dose was raised by adding ♏j alternate days until ♏viij were reached and then it was omitted for three days.

On April 4th pil. ferri. gr. v was given in addition twice a day.

Recovery was necessarily very slow.

Blood-count.—(Fig. 3, No. 3, May 16th.)

On April 18th, Dr. Embleton estimated the fragility of the red cells *in vitro* and found that they were more resistant than normal.

The numbers were: Complete hæmolysis in 0·25 per cent. of NaCl; slight in 0·4 per cent. and *nil* in 1·00 per cent. No bile was found in the serum.

During the period of recovery the mouth and tongue showed superficial ulceration, but we must add that arsenic had been given for some considerable time before this.

The patient left the hospital at the end of May, and a month later returned to report herself. She had kept well, but had gained but little strength. Her general appearance was no worse; her heart was as before. The spleen was palpable, the liver could not be felt. Her knee-jerks were just obtained.

Blood-count.—(Fig. 3, No. 4, June.)

Unfortunately the journey was undertaken upon one of the few really hot days in the summer, and she overtired herself. From that date evil symptoms arose again, and were of the same nature as before, namely, increasing weakness, dyspnœa and anorexia, followed by vomiting.

Readmitted on July 26th, she was even more yellow than on the first occasion; there had also been some pain over the heart, and swelling of the ankles. All ulceration of the mouth had disappeared. Her speech had again become slow. The temperature was raised, and remained so, as the chart demonstrates (Figs. 4 and 5).

We will not weary you with a repetition of the symptoms of the general state, but dwell upon the salient points. (*Blood-count*, Fig. 3, No. 5). The spleen was now considerably enlarged and tender, but the liver was not palpable at first, though later it enlarged and also became tender. Delirium and

Blood Counts

Mrs M.

			Red Cells per c.mm.	Hb %	Colour Index	White Cells per c.mm.	Lympho-cytes L	S	Polymorphonuclear Neutrophil	Oxyphil	Basophil	Myelocytes	Hæmatoblast	Transitional	Remarks.
1	March	21	980,000	20	1·02	15,750	13	31	50	0	0	5	0	1	Poikilocytosis, Anisocytosis &
2	April	12	1,170,000	35	1·34	15,400	10	25	59	6	0	0	0	0	vacuolation were present
3	May	16	2,870,000	65	1·05	35,200	22	16	59	1	0	0	1	1	throughout
4	June	26	2,600,000	25	·85	18,700	6	19	72	2	1	0	0	0	Average size of red cells greater
5	July	27	1,940,000	22	·6	10,500	24	12	63	1	0	0	0	0	than normal.
6	August	2	1,180,000	22	·8	3,850	13	32	55	0	0	0	0	0	Nucleated Red Cells first seen
7	"	20	986,000	25	1·1	5,250	7	25	66	5	0	0	5	1	Stippling and polychromatophilia
8	"	27	1,080,000	30	1·4	4,900	15	15	66	0	2	1	0	1	Giganteblasts seen

FIG. 3.—Specimens from the blood-counts. The horizontal line terminating on the left in a star divides those made in the first illness from those in the second.

a curious aloofness of mind, which she herself recognised, reappeared, but restlessness was not present. This phase, which lasted ten days on the first occasion, lasted about nine days this time. The peptones a trace; no free HCl; no mucus. Total acidity = ·03467 per cent. HCl; total chlorides = ·0292; digestive power = 37 per cent.

Dr. Thursfield was good enough to estimate the

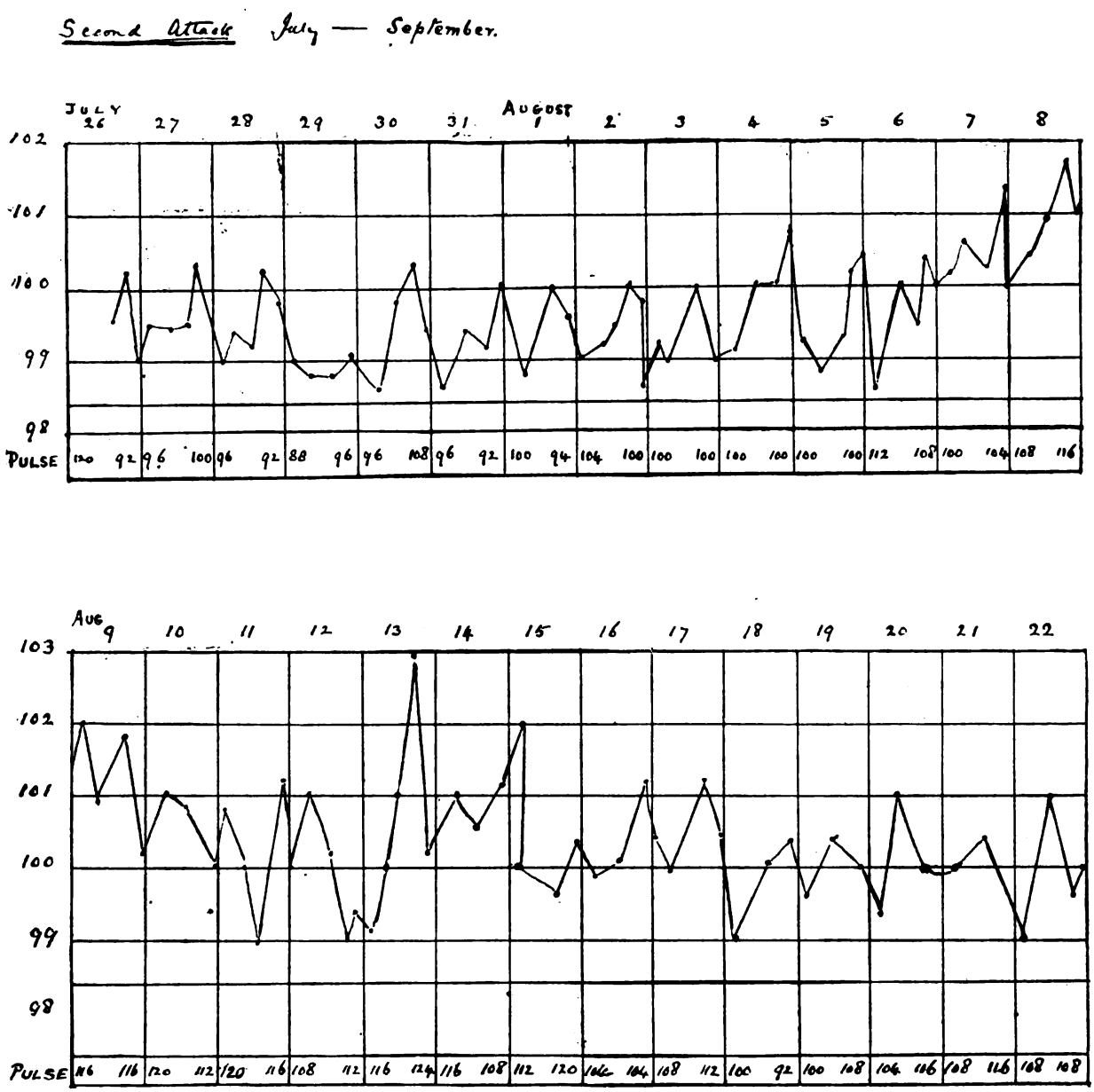

FIG. 4.—The temperature chart in second illness.

blood-count on August 2nd indicated a blood crisis (*blood-count*, Fig. 3, No. 6).

The fæces during the attack became a little pale, but never acholic. Dr. Embleton examined them for worms and ova with negative results. The urine remained a natural colour.

On this occasion the gastric juice was examined with the following result: Lactic acid present; fragility of the red corpuscles in this attack, and confirmed the result obtained by Dr. Embleton in the first illness, which demonstrated an increased resistance of the red cells to sodium chloride *in vitro*.

On August 14th 0·4 grm. of neo-salvarsan was given intravenously, and this was repeated on the 29th. We do not think this treatment produced

any good effect, and it would seem from a survey (*blood-count*, Fig. 3. No. 7) of this case while under observation that neither Fowler's solution nor neo-salvarsan was certainly helpful. The impression we gained was that the disease ran a tolerated in the smallest doses, and her tongue and mouth have become sore. The temperature has risen, and the knee-jerks are lost, but her mind is fairly clear. It appears to us that the fatal issue is imminent.*

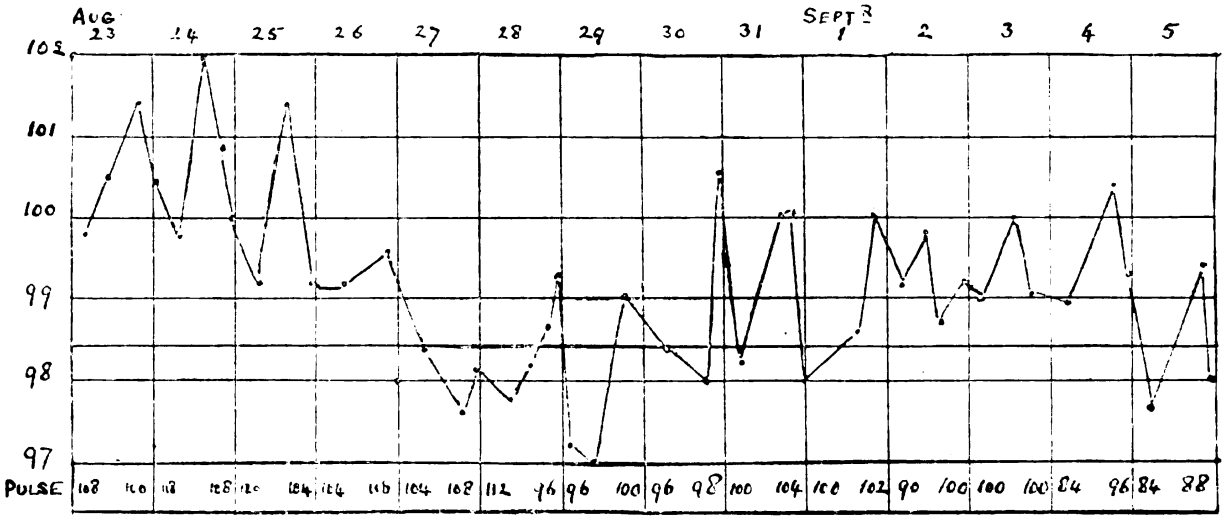

FIG. 5.—The temperature chart in the second illness (*continued*).

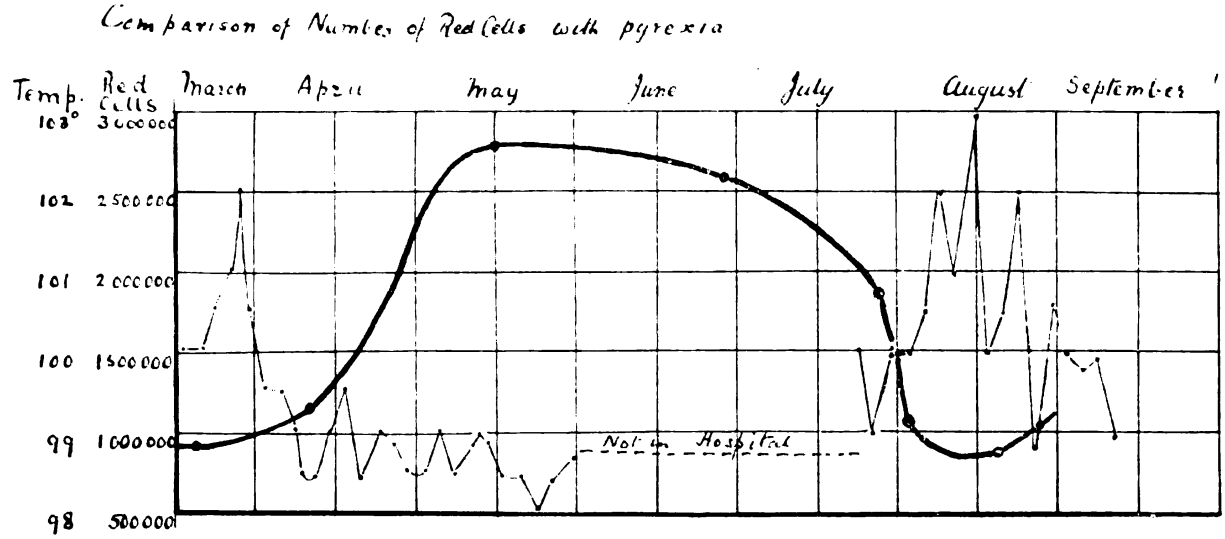

FIG. 6.—A diagram showing the relation of the temperature chart to the red cell count. The heavy dark line represents the varying red cell count.

cycle of its own, irrespective of those drugs (*blood-count*, Fig. 3, No. 8).

On September 5th a bruise was noticed on the dorsum of the left hand, but no retinal hæmorrhages have been detected.

During the latter part of September, after a period of improvement, there has been another relapse. Dulness, yellowness and weakness with dyspepsia have reappeared. Arsenic cannot be

We have, then, here, the history of a virulent anæmia with acholuria; with enlargements of the liver and spleen which vary with the course of the illness; with fever, which is remarkably persistent,

* Once again a marvellous recovery has occurred, and the patient, slowly improving, left hospital just before Christmas, with a curiously bright red coloured complexion. She remains at present well.

and which varied inversely with the red blood-count as this chart shows.

The changes in the blood are extreme. The red cells are greatly diminished in number, the index is high. The blood-corpuscles are much altered with a tendency to increase in size ; there marking upon the striking effect of over-exertion upon this illness, and also upon the remarkably acute onset of the periodic attacks. These certainly demand some explanation if the view is taken that there is some local focus of infection at the bottom of this illness.

[Dr Poynton's Cases]

Recurrent Familial Jaundice.

	Red Cells	Hb %	Colour Index	White Cells	LL	SL	Polynuclear Neutr.	Ox.	Ba.	Myelocytes	Hyaline	Transitional	Remarks.
Ada C.	3672000	56	·7	13576	10·2	28·9	53·3	3·5	0	0	0	4	Normoblasts present
	2982000	64	·97	13575	42·3	12·6	36	4·5	0	0	0	3·6	
* Albert C.	1904000	46	1·1	28280	4·9	48·3	44·3	1·2	1·8	+	·4	0	* note low red Cell
*	1720000	28	·81	13700	8·6	12·6	75·3	2·0	0	0	0	1·3	* Count.
	2084000	36	·9	13126	10	41	37	0	2	+	10	0	
Ivy C	2200000	52	1·18	11700	4·3	2·6	46·6	4·3	1	+	0	1·3	Poikilocytosis, Anisocytosis, Vacuolation, Polychromatophilia,
	3285000	60	·8	24100	7·6	27	58·1	3	1·3	0	3	0	nucleated Red cells.

FIG. 7.—Blood-counts. Congenital acholuric jaundice.

[Dr Dodgeon's Cases]

Congenital Family Cholaemia

	Red Cells	Hb %	Colour Index	White Cells	LL	SL	Polynuclear Neutr.	Ox.	Ba.	Myelocytes	Hyaline	Transitional	Remarks.
Case I.	3676000	70	·9	6120	23	6	82·2	·6	0		9·8	·6	All cases showed poikilocytosis
" II.	3043000	35	·58	3800	6·5	23·2	64	·75	0		3·7	·25	and bile stained serum
" III.	3038750	35	·58	4160	11	23·2	57·5	1·5	0		6·2	0	Polychromatophilia & Anisocytosis —X
" IV.	2593000	35	·7	5440	Differential not given								Normoblasts and megaloblasts. —X

[Dr Parkes Webers Case]

Acquired Chronic Acholuric Jaundice.

	Red Cells	Hb %	Colour Index	White Cells	Lympho Cytes	Polynuclear Neutr.	Oxy	Ba	Myelocytes	Hyalines	Enkrmedale	
* — Jan 31	900000	18	1	6000	45·6	46	·8	0	0	2·4	4·8	Poikilocytosis, Polychromatophilia,
May 19	2000000	35	·8									Erythroblasts, mast cells
Aug 15	1300000			8200	51·2	37·6	·4	0	+	1·2	9·6	Myelocyte seen
Dec 8	3277000	70	·9									
Dec 21	4123000	74	·6	6250	26·6	68·4	·8	0		2	2·2	No abnormality.

FIG. 8.—Blood-counts. Congenital acholuric jaundice and acquired acholuric jaundice.

has been one definite blood crisis. The fragility is, however, not greater, but less than normal. A considerable leucocytic increase has given way to a leucopenia. Digestive disturbances have been prominent throughout, as also mental symptoms and diminution of reflexes.

We have found no septic focus, though its possible existence cannot be gainsaid ; even if it were obvious the practical physician could not help re-

It is in the face of these facts that we venture now to compare some of the features in this case with those that occur in congenital family cholæmia, recurrent familial jaundice, or congenital acholuric jaundice, whichever term may be preferred. Some of the earliest examples of the disease in this country were shown here by one of us in 1906, and some of the Fellows may perhaps remember them. In these cases we find sometimes

a persistent yellow colour, or, again, a curious pallor varied by attacks of yellowness. After an attack of increased yellowness there may be considerable anæmia (*vide* chart). The liver and spleen are enlarged and vary with the phase of the disease. There may be outbursts of fever, which may in young children be persistent and considerable. There may be tenderness and pain over the liver or spleen. The blood shows definite changes; the red cell count in one of the cases fell to under two million, and Dr. Guthrie recorded, we believe, one in which the count was about a million. There are poikilocytosis, anisocytosis (Figs. 7 and 8) and vacuolation. The colour index is often high. The fragility of the red blood-corpuscles is greater and not less than normal; but is this a constant feature? Here is a point upon which we should like information. The urine is almost invariably free from bile-pigment, and the fæces of good colour, though sometimes rather pale. In four of these families under observation no mental disturbances have been noted during attacks. How alike even in some details are such cases as these to the one recorded here, yet how different in their practical meaning! These congenital cases, though delicate and maimed by their condition, live for years in active employment. Our case to-night, on the other hand, shows every sign of advance to a fatal termination after a comparatively short illness.

The point we wish to raise to-night is, Where does the essential difference lie? Are we to see in the laboratory test of the fragility of the red blood-corpuscles a vital distinction? The congenital origin of the one and the acquired origin of the other is bridged over by such cases as have been published on the continent and by Dr. Parkes Weber in this country under the title of "acquired chronic *acholuric jaundice*." The congenital cases can hardly be dependent upon some local focus of sepsis using this term very generally, and probably the balance of evidence favours some inherent fault in the blood-forming organs. Are we to consider such a case as the one we record to be dependent upon some such local focus, or upon an inherent fault in the blood-forming organs? Lastly, what importance are we to attach to the recent view of Pappenheim that the hyperchromatosis in pernicious anæmia is not due to the augmentation of the hæmoglobin in the red corpuscles but to a degenerative condition of the colouring matter?

We know that we may have omitted many points in the difficult investigations of this case, but if we succeed in obtaining the views of others upon these questions the object of our communication is served.

Discussion.

Dr. Box said he had been connected with cases of the same nature as that which had just been described, and he would give particulars of a case which in many respects was similar. The patient was admitted under Mr. Corner for gall-stones, and he asked him, the speaker, to see the case from the point of view of diagnosis. He was a man, æt. 50 years, a platelayer. There was nothing important in his previous history, but in January, 1911, he became yellow, and began to suffer from pain in the right subcostal region, which latterly extended to the cardiac area. It was particularly present on exertion, but disappeared during rest. He also had attacks of palpitation, and noticed increasing asthenia, which led him to consult a doctor on three separate occasions. The depth of the jaundice varied from time to time. During the last three months he had had swellings of the legs, and was sent to the hospital on account of the recurrent pain in the right subcostal region. When he examined the man he was very yellow, so that it was thought it might be obstructive jaundice; the jaudice involved both skin and conjunctiva. The heart was dilated, and there were hæmic murmurs. The liver was a little enlarged, and the spleen was palpable. The urine was clear and acid, sp. gr. 1015. It contained excess of pigment, and there was no bile, albumen, or blood. On further inspection the mucous membranes were seen to be unnaturally pale, and the blood examination showed him to be suffering from megaloblastic anæmia, leucopenia and relative lymphocytosis, with a red cell count of 1,056,250. The colour index was 1·3, hæmoglobin 30 per cent., serum yellow. On the strength of that the diagnosis of pernicious anæmia was made, with probable cholæmia. On September 10th he was given an intravenous injection of ·6 salvarsan, and that was followed by a rigor and vomiting, and a rise of temperature to 101·5° F. For a short time after the injection the yellow colour was deeper; afterwards it became paler, and ten days later the count was 468,750 more reds, colour index 1·1, the white cell count being very little different. At a count seven-

teen days later the red cells had fallen to 1,206,250. Another injection of salvarsan was given, and he again became yellow. A blood-count ten days later showed a slight improvement to 1,212,500, colour index 1·2. There was still leucopenia and relative lymphocytosis. The patient was at present at the Convalescent Home, but probably he would soon be back.

The second case was that of a little girl with congenital family cholæmia. He first saw the mother in 1884, when she was under Dr. Bristow, and after that she was admitted again on various occasions and with various diagnoses. Dr. Bristow said it was anæmia with enlarged spleen; Dr. Thorne's diagnosis was leucocythæmia. Later still the diagnosis of Banti's disease. She came under his own observation again eight or nine years ago, bringing one of her daughters, who had an enlarged spleen and a yellowish coloration of her skin very similar to her mother's. That led to an investigation of other members of the family. Three or four children had enlarged spleens, but this girl was the only member who had cholæmia. She was now 12 years of age, and the salient point was that she suffered, and had recently suffered very much, from crises of abdominal pain and vomiting, with collapse, and that had made her practically an invalid for four years. He got Mr. Corner to see the case, and after consulting the mother it was decided to excise the spleen. For three or four days after the operation she was very well, except for some fever, with a temperature reaching 105°F. But then the temperature sank to normal. Then she vomited and collapsed, and death was expected, the yellow colour disappeared, and she was now fairly on the way to convalescence. Before the excision of the spleen the red-cell count was 3,337,000 (?), hæmoglobin 69 per cent., colour index ·9, leucocytes 10,640. The differential count showed 50 per cent. polynuclears, 40 per cent. small lymphocytes, 3·75 per cent. large lymphocytes, 3·07 hyaline cells, ·5 per cent. myelocytes; 5 normoblasts present in the field. A week after the excision of the spleen, the reds were 4,950,000, hæmoglobin 80 per cent., colour index ·8, leucocytes 6220, and there was a slight increase of polynuclears, which was probably due to suppuration in the superficial tissues.

Dr. PARKES WEBER said that, but for the lateness of the hour, he would have been tempted to remark on the different kinds of acholuric jaundice, but he would be brief, and practically content himself with thanking the authors for their interesting communication, which furnished a valuable datum on the subject. In this case, considering the pyrexia, the enlargement of the spleen and liver, the anæmia, and the ultimate development of the leucopænia, he would be inclined to class the case as one of septic infection. He believed it was possible to meet with all those symptoms in a case of chronic endocarditis, maligna tarda as it was known on the continent. Sometimes the disease was not recognised during life, even by very good men, as one affecting the valves of the heart. Still, such cases did not always affect the valves of the heart. He had seen one case, with symptoms rather different from those in this case, in which, after long-continued pyrexia, the lesion which was found was a chronic endarteritis of the first part of the pulmonary artery, but beyond the valves. He thought it possible that cases of the same class existed in which neither the heart nor the large arteries showed any signs of endocarditis or endarteritis after death, and in which the septic disease was in more distal parts of the circulation.

Dr. FREDERICK TAYLOR said he had now under his care at the Seamen's Hospital, Greenwich, a case which seemed to present exactly the features of pernicious anæmia and acholuric jaundice. The patient had been under his care since March, and when she first came in it was obvious that she was anæmic, and yet the yellow tinge was distinctly mixed with green, so that she was obviously the subject of icteric yellowness, and that she had not merely the lemon-yellow which was characteristic of uncomplicated pernicious anæmia. During her stay this biliary tinge had persisted, and throughout he had felt it was not a case of uncomplicated pernicious anæmia. He had not provided himself with the documents which would enable him to give to the meeting the numbers of blood-cells, etc., but until August her blood-count was never more than 1,600,000. When she was first admitted the number was 1,400,000, and within ten days it had fallen to 700,000. In that interval there had been a high colour index, varying from 1·2 to 1·6. He believed that at first there was leucopenia; there had never been marked leucocytosis, but the count of leucocytes had sometimes been above the normal. There had been a marked biliary tinge in the skin but the urine had not contained bile. There had not been marked pyrexia, no such temperature as in Dr. Poynton's case. The spleen had been definitely enlarged throughout the illness, always 1½ to 2 inches below the costal margin. She was a young

woman, of somewhat small stature. She was treated at first with arsenic, but it seemed to make no impression upon her; he did not think it agreed with her. A long time ago his house-physician was anxious to have her injected with salvarsan. He, Dr. Taylor, tried her with bone-marrow for some time, but there was no improvement. So in the month of August she was injected with ·2 grm. of salvarsan, and a week afterwards there was the greatest increase in her red blood-corpuscles that there had ever been, as they numbered now something over 2,000,000. A second injection in that month was followed by another slight rise. She had a third injection a fortnight ago, but he did not remember what effect it had had. She still had a distinct jaundice tinge, but the appearance of the mucous membrane was now more favourable than formerly. She certainly looked better generally, though perhaps part of her anti-anæmic look was due to the yellow coloration. She always protested that she was better, she had not wasted, and did not appear to be very ill. What the outcome of the case would be he could not say, but he was glad to receive the hint from Dr. Parkes Weber that perhaps it was not pernicious anæmia, but septic poisoning. He did not think it was malignant endocarditis, but in view of the uncertainties of that disease he would not say it was not so until later on. Some time he might be able to publish the case in a more complete form.

Dr. GORDON R. WARD asked whether the colour of the fæces in Dr. Poynton's case varied during the exacerbations of the cholæmia of congenital origin. He believed that in some of the acquired cases the fæces had been much more highly coloured when the patient had the severe attacks. With regard to the case narrated by Dr. Box, he thought it was a good thing that operation had been performed in this country for such, and he hoped the case would be published complete, because early cases, although successful, did not seem to have received the notice they ought. He knew of only two, and they were ten years ago.

Dr. POYNTON, in reply, thanked the Fellows for discussing the case. Not being alive to the point, he could not properly answer Dr. Ward's question about the colour of the fæces, but in the second illness, the present one, while the temperature was high, the fæces was lighter in colour than during the first attack, when the temperature was not so high. Dr. Box's case was most interesting, and he shared the hope that it would be published in detail. He was also interested in Dr. Parkes Weber's remarks,. because he had been good enough to see the case twice for him. If there should be an opportunity, he would report further upon the case.

February 3rd, 1913.

ON THE TREATMENT OF CLUB-FOOT IN EARLY CHILDHOOD.

(Based on a lecture-demonstration given at the Queen's Hospital for Children on December 10th, 1912.)

By A. S. BLUNDELL BANKART, M.C.Cantab., F.R.C.S.Eng.

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GENTLEMEN,—Club-foot, or congenital talipes equino-varus, is *curable* in infancy and early childhood; that is to say, in the majority of cases it is possible to convert the deformed foot into one which is almost indistinguishable from normal, which is supple and capable of good movement, and which retains its shape ultimately without support of any kind. At a later age—indeed, at any age—it is always possible to obtain a foot which is plantigrade and fairly useful—that is, one which is placed at right angles to the leg and with the sole coming squarely to the ground. But although that will enable the patient to walk, it is often at the expense of much of the foot's mobility, and the result cannot be compared with what might have been obtained by treatment at an earlier age. So to do justice to cases of club-foot they must be treated in infancy, and this should always be possible inasmuch as practically every case is seen by a medical man at, or soon after, birth, and the parents are always anxious that something should be done at once. Treatment should be begun immediately after birth.

Congenital talipes equino-varus is a deformity which involves a number of joints, each of which contributes a part to the general distortion of the foot. The condition is complicated only by the fact that the joints are small and placed very close together, so that their several deformities appear to be blended into one. Each of these lesser deformities consists of a distortion of a joint in the direction of one of its normal movements—it may be within the limits of normal movement, or it may be grossly exaggerated—so it is to the normal movements of the joint that one must look in order to understand its deformity.

Now, if you take any joint and bend it into any position, either a normal one or an exaggeration of

a normal one, then, of course, the soft parts are relaxed on the side towards which it is bent, and on the opposite side they are stretched. Next, suppose the soft parts—skin, fascia, muscles, tendons and ligaments—adapt themselves to the position in which the joint is held; on the concave side the parts which were relaxed become shortened, while on the convex side the stretched parts are permanently lengthened. Thus the joint is fixed in that position and movement in the opposite direction is resisted by the shortened structures. Lastly, suppose this condition has existed for a long time, or that it has arisen during the development of the part, then you will find that the bones themselves are adapted to the deformity, they do not fit in any other position, so they naturally assume that position when left to themselves. This, anatomically, is the type of deformity with which you have to deal in club-foot, and you will observe that three factors determine the deformity in each situation: (1) a faulty position at a joint; (2) structural shortening of the soft parts, especially the tendons and ligaments, which would otherwise be relaxed in that position; and (3) alterations in the shapes of the bones.

It is usual to divide the deformity of equino-varus into two parts, namely, equinus, which occurs at the ankle-joint, and varus, at the medio-tarsal joint. But varus is a good deal more than a simple deformity of the medio-tarsal joint. Varus itself is composed of two parts: (1) a distortion at the medio-tarsal joint; and (2) a distortion at the subastragaloid joint.

At the ankle-joint the foot performs simple movements of plantar flexion and dorsiflexion. Fixation of the foot in any degree of plantar flexion is talipes equinus. In this deformity the heel is drawn up and fixed by the shortened tendo Achillis and posterior ligaments, the toes are pointed downwards, and the front part of the body of the astragalus is tilted forwards and projects from between the malleoli.

At the medio-tarsal joint (astragalo-scaphoid and calcaneo-cuboid joints) there is a gliding movement inwards of the scaphoid and cuboid upon the head of the astragalus and the anterior end of the os calcis respectively, so that the front of the foot is directed inwards. But, owing to the obliquity of the calcaneo-cuboid joint, the cuboid does not glide straight inwards, but a little downwards as well as inwards. This introduces a movement of rotation which causes the outer border of the foot to be depressed so that the sole is inverted. Fixation of the foot in this position, or in an exaggeration of it, is one part of the deformity of varus, and the structures which are chiefly concerned in fixing it are the two tibial tendons (tibialis anticus and tibialis posticus) and the ligaments at the inner side of the foot (especially the astragalo-scaphoid and the anterior part of the deltoid or internal lateral ligament of the ankle).

At the subastragaloid joint the os calcis rotates beneath the astragalus about a vertical axis which passes through the interosseous astragalo-calcanean ligament. This axis is situated at the inner side of the body of the os calcis, between it and its lesser process, so that, in rotating about it, the body of the os calcis glides forwards beneath the astragalus, and its anterior extremity is carried forwards and inwards. This, of course, pushes the cuboid and with it the outer metatarsal bones forwards and inwards, so that the front of the foot as a whole is directed inwards, *i.e.* adducted. But here, again, owing to the obliquity of the joint between the body of the os calcis and the astragalus, the os calcis does not glide straight forwards, but also a little downwards. This in turn depresses the cuboid and outer border of the foot, so that the sole is inverted. Fixation of this position of adduction and inversion, or an exaggeration of it, at the subastragaloid joint, constitutes the remaining part of the deformity of varus, and the soft parts chiefly concerned are, again, the two tibial tendons, and the interosseous astragalo-calcanean ligament.

Varus is, therefore, a compound of medio-tarsal and subastragaloid displacements, the combined effects of which produce adduction and inversion of the foot, so that the toes are directed inwards; the outer border of the foot is rounded and depressed, the inner border is concave and drawn up towards the leg, and the sole looks inwards and backwards or directly backwards. Equino-varus is the same deformity with the added one of equinus at the ankle-joint, and this is the condition which has to be unfolded in the treatment. You will see in this specimen (skeleton of club-foot shown) how not only are the bones displaced in the directions I have indicated, but

how they are actually altered in shape and moulded to the deformity so as to fix the foot in that position. The most striking individual change, perhaps, is the inward and sometimes downward deflection of the head and neck of the astragalus, but to some extent practically every bone in the tarsus is affected.

Now, you will see at once how impossible it is to correct such a complicated deformity by any localised operation on the bones. It is true that by removing sufficient bone the foot can be brought into an approximately correct attitude, but there always remains some part of the displacement untouched, and experience has shown that if a so-called "radical" operation is relied upon as the sole means of treating club-foot, the deformity invariably relapses as soon as the patient begins to walk about. Drastic operations are reserved for older cases in which other means have failed; they do not cure the deformity in the true sense of the word, but they effect a compromise by providing a more or less stiff foot in a correct attitude; moreover they are not final, but they have to be followed by the same long after-treatment as any other method.

The correction of club-foot in young subjects is brought about by tenotomy and manipulation. When you attempt to put such a foot into a correct position you will find that the skin and superficial parts are easily stretched; the first serious resistance is that of the contracted tendons, which will have to be divided; the faulty position is then maintained by the shortened ligaments, against which your manipulation is directed. If the child is very young, frequent manipulation of the foot towards the correct position will stretch the ligaments, and if each improvement is maintained by a splint in the intervals, the faulty position will be gradually overcome. In older patients you may have to use great force to tear the ligaments, or you may have to divide some of them with a tenotomy knife. In one or other of these ways the resistance must be overcome until the foot can be placed easily in an over-corrected position.

But club-foot is not cured when the position of the foot is corrected, because, you will remember, the bones are adapted to the deformity, and if they are left to themselves they will inevitably fall back into the old position and the deformity will recur. If, however the foot is kept in the over-corrected position for a sufficient length of time, the bones will adapt themselves to the new position, they will become altered in shape to suit it, and in time they will retain it naturally of their own accord and without artificial support of any kind.

This is the principle upon which the treatment of club-foot is founded—over-correction of the displacement at the earliest possible age, and retention of the foot in the correct position until the correction has become natural and permanent.

It is customary to divide the correction of equino-varus into two stages—first, to correct the varus, and when that is fully corrected, to deal with the equinus. The reason for this is, that when the foot is in the equinus position it is easy to apply a straight splint to the outer side of the leg and foot, but when the foot is at right angles to the leg it is very difficult to get any purchase on the front of the foot.

In the correction of varus the first step is to divide subcutaneously the tendons of the tibialis anticus and tibialis posticus, and I should advise that this always be done, even if they do not appear to be very tight, because you may be sure they are too short, and in any case they tend to reproduce the deformity whenever they act. Some of the most difficult relapsing cases are those in which the tibial tendons have not been divided at the beginning of treatment. As soon as they are divided, a small gauze dressing is placed over the punctures and a straight "malleable iron" splint is applied to the outer side of the leg and foot, from the knee to the toes. At first the lower end of the splint may be bent in a little to adapt it to the position of the foot. After a few days manipulation is begun, the splint is removed, the malleoli are grasped with one hand, while with the other the front part of the foot is firmly manipulated outwards, outwards and upwards, and with a twist towards eversion. This is repeated many times, and then the splint is firmly bandaged on, its lower end being now bent outwards as far as the correction of the foot will allow. It is very important to put on this splint correctly. First, bandage lightly up the leg, then catch and fix the upper end of the splint with a turn of the bandage; next bandage from without inwards down the leg and splint, and as the foot is reached draw it firmly outwards, catching it to the splint with each turn of the bandage. See that the toes do not go blue;

if they do, the bandage must be loosened and the correction diminished.

It is usually recommended that the heel be firmly held while the front of the foot is manipulated, but a glance at the anatomy of club-foot will show you that that is wrong. When the heel is fixed, the manipulation falls entirely on the medio-tarsal joint, but that, as we have seen, accounts for only a part of the varus displacement. In order to correct at the same time the subastragaloid displacement the os calcis must be free to rotate, and then the manipulation acts on both parts of the deformity. Fortunately, in an infant with club-foot it is practically impossible to fix the diminutive heel effectively, and the result is very good, but the older the child the more easy is it to grasp and fix the heel, and so, proportionately, is the result less satisfactory. As a matter of fact, the correct application of the splint is as important as, if not more important than, the manipulation of the foot, and in hospital practice we find that better results are obtained when the child is brought three times a week to have the foot worked and the splint reapplied by a trained nurse than when the parents are allowed to remove the splint at home in order to work the foot. You will notice, of course, that the splint cannot possibly fix the heel. In this way the varus deformity may be completely corrected in about a month, and then the foot is in the position of simple talipes equinus. The test of completeness is that the child can voluntarily evert the foot, and you should not be content until that is done.

The next step is the correction of the equinus deformity at the ankle-joint. But before doing this it is better to wait a few months until the child has grown a little, because then it is easier to control the foot in a splint. Equinus is corrected by dividing the tendo Achillis and at once forcing the foot up into a position beyond a right angle with the leg. This position must be maintained by a splint and at the same time the varus correction must be kept up. For this purpose you may use a simple tin-shoe or splint (such as this) with the foot-piece fixed at a little less than a right angle; it is kept on with a bandage. But with this you will find it rather difficult to control the foot, and a much better, though slightly more complicated, appliance, is Adams' varus splint, which I have here. In this the foot-plate is attached by a screw-rack, so that the angle it makes with the leg can be altered to any extent. Across the ankle runs a thickly padded strap to keep the heel down, and the leg and foot are secured by webbing-straps. You will notice that it also extends to the thigh, so as to give greater security. To put it on, the foot-plate is screwed down a little, and the heel is firmly fixed upon it by the padded strap. The leg being secured, the foot is racked up to beyond a right angle with the leg, and then the strap round the front of the foot is fastened. This last strap passes through a wire loop on the outer side of the foot-piece, and it is arranged so that it draws the front of the foot outwards. This instrument keeps the deformity fully corrected, and, except when it is removed for manipulations, it should be worn day and night until the child is old enough to walk.

At about the age of two years you will generally find that the child is scrambling about on his splints and breaking them up, and then you will have to order a walking instrument. Such an instrument must be worn for some years if a permanent cure is to be obtained, and there must be no shirking of either the trouble or expense, for you may take it as an absolute certainty that the foot will relapse if it is allowed to go unsupported before the moulding of the bones is complete.

Now, walking instruments, though rather complicated at first sight, are really quite simple in design. Here is a boot with a T-shaped strap fixed to the outer side of the sole; and on the inner side there is a slight steel bar, jointed opposite the ankle and fixed below into the sole of the boot, while above it is attached to a band which goes round the calf of the leg. When it is put on, the free ends of the T-strap are fastened round the leg *and bar* just above the ankle, and on drawing them tight the outer side of the boot is tilted up and the sole everted. This simple contrivance, then, tends to keep the foot everted while the child walks.

But if you examine a case of club-foot which has been corrected you will find that when the patella looks straight forwards the foot is almost invariably directed inwards. This is not due to any fault of the foot, but it is due to a twist in the leg, and the result is that the child walks with the toes turned in, even though the club-foot deformity is wholly corrected. To some extent this internal rotation can be diminished by repeatedly twisting

the leg outwards whenever the foot is manipulated, but when the child begins to walk the turning-in of the foot must be prevented or the foot will certainly relapse. The only way to do this is to extend the instrument right up to the pelvis. A second steel bar is attached to the outer side of the calf band and carried from there upwards, first to a band round the thigh and then to a band round the pelvis. A joint is put opposite the hip. By adjusting the attachment of this bar to the pelvic band the whole limb can be kept rotated a little outwards. Some surgeons use this single inside bar below and single outside above, but I much prefer to have the instrument double right up the leg and thigh. This not only gives extra strength (instruments are very liable to break when the child is active), but it gives better control over the whole limb and especially over the knee. Instrument-makers always put a joint opposite the knee, but when the knee is bent it is impossible to control the rotation of the leg, and you will find that the child always walks with the knee bent and the foot turned in. Therefore, always see that the joint opposite the knee is fixed by a pin or rivet driven through it and make the child walk with a stiff knee. The knee is kept in place by one or two knee-caps. The complete instrument, then, is as follows: Boot with varus T-strap, double uprights to thigh, single to pelvis, pin at knee and double knee-caps. Lastly, a simple tin shoe must always be worn at night.

Now begins a period of supervision which will extend over several years. At first you will see the child fairly frequently, say once a month or six weeks; then, if all goes well, once in three months; and finally twice a year. Your duty will be to see that the correction of the deformity is maintained, that the instrument is efficient and put on properly, and that it is kept in repair and adjusted to the growth of the child.

In hospital practice one often finds that the foot begins to go back a little when the child first starts to walk. When this happens it should be taken in hand at once because the tendency is always to relapse, and it will never correct itself. The child should be given an anæsthetic and the foot should be forcibly wrenched with the hands into an over-corrected position. Then it should be put back into the tin shoe for two or three weeks, after which the child may begin to walk again in the instruments. The foot should still be manipulated every day at home.

The treatment which I have described to you is the routine treatment which we employ in straightforward cases in early childhood. There are, of course, other methods which aim at a more rapid correction of the deformity, but the basis of permanent cure is the remodelling of the skeleton during growth, and this can only be established in the course of years. Gradual correction gives a more perfect foot than any other method of treatment. There is no form of treatment which can dispense with the necessity for long after-treatment and instrumental support. Unfortunately, orthopædic hospitals are still crowded with the results of neglected after-treatment, and the most fearful and intractable relapses are those in which drastic bone operations have been undertaken in the mistaken belief that they will cure the deformity once and for all.

Of the older and more difficult cases I shall not have time to speak to-day; I will only ask you to remember that they ought to have been treated in infancy.

February 3rd, 1913.

Operative Treatment of Pelvic Phlebitis.—Vanverts and Paucot tabulate the details of 82 cases of operative treatment of puerperal thrombosis, with recovery in 33, as reported by 26 writers. Lenhartz and Bumm each have reported 8 cases, including 4 recoveries. Study of the tabulated details suggests that utero-pelvic phlebitis requires treatment to prevent escape of emboli and transmission of infectious products through the veins, while the original phlebitic focus should be directly attacked. In practice, however, the former is the most important, and the original focus may sometimes be left untouched. If the veins can be ligated above the thrombosis this should be done, and if there is suppuration in the vein or an abscess in the broad ligament, the abdominal incision can be closed and the purulent focus opened and drained by extra-peritoneal access, either through the vagina or through an incision above Poupart's ligament. If the pelvis shows no signs of suppuration or there is merely slight infiltration along the vein, it may not be necessary to attack the focus directly. If symptoms of suppuration in the broad ligament develop a few days later, the focus can be opened then below the peritoneum.—*Journ. A.M.A.*, vol. lx, p. 90.

CLINICALIA.

Extracted from the CLINICAL JOURNAL.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from p. 272*).

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The Restitution of Function the most important Point in the Treatment of Fractures.

Though I am quite alive to the value of X-ray evidence in helping us to decide on the right line of treatment and in controlling the anatomical result, I am sure it is important to realise that it gives no indication of the functional result, and that on the basis of restitution of normal function both non-operative and operative treatment of fractures must be judged.

("The Selection for Early Operation of Fractures of the Long Bones," by C. H. Fagge, M.S., F.R.C.S., 'Clin. Journ.,' vol. xxxix, p. 385.)

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The Diagnosis of Duodenal Ulcer,
or
The Modern Craving for Concentrated Foods, Mental as well as Physical.

During a discussion on duodenal ulcer Mr. Moynihan remarked—"Duodenal ulcer was a real disease. It could be recognised at operations and could be demonstrated, and it could be diagnosed. And he wished to say deliberately that it could be diagnosed with an accuracy which was not exceeded in the case of any other abdominal disorder. But in making a diagnosis every detail must be gone into with the most sedulous care. It seemed to be one of the faults of modern medicine and surgery that one must deliver one's message in the most concentrated form; one was expected to describe the torrent of Niagara in terms of the pipette. He had been held to describe all the aspects of duodenal ulcer in the single term 'hunger-pain.' The term 'hunger-pain' as he originally described it meant duodenal ulcer, and meant nothing else. . . . If the term was taken in the meaning which he originally attached to it, he was still confidently of opinion that it meant a duodenal ulcer. If the hunger-pain was subject to the seasonal variations which he had described, if it was recurrent, if it occurred at particular times, and was relieved by particular means, then in his belief it was applicable only in cases of duodenal ulcer."

("Discussion on Duodenal Ulcer," 'Clin. Journ.,' vol. xxxv, p. 391.)

(151)

"What I have Said, I have Said,"
or
The Folly of always adhering to one's Original Diagnosis.

Later on Mr. Moynihan said: "Some surgeons seemed to think that it was a humiliation for them to open an abdomen, after making a diagnosis on reasonable grounds, and not to discover the particular condition which they had supposed to be present. He had himself no compunction in throwing his diagnosis aside as soon as it was found to be wrong and going in search of something else. And if any precedent was required it could be found in the example of Saul, who went in search of his father's asses and found a kingdom.

* * * *

The one thing against which he could not inveigh too strongly and too constantly was that of making the diagnosis, not verifying it at the operation, but proceeding, nevertheless, to the performance of the operation originally planned.

(*Ibid.*)

(152)

An Essential to the Success of Vaccine Treatment.

When the infection has been ascertained the question of a vaccine comes to the front. Vaccination is good when done under proper circumstances and preceded by a correct clinical diagnosis. Without a clinical diagnosis vaccination is mere scientific quackery. Good results are not likely to ensue when a pyosalpinx has been overlooked or an appendix full of pus.

("With Mr. Lockwood in the Wards of St. Bartholomew's Hospital," 'Clin. Journ.,' vol. xxxix, p. 198.)

(153)

The Best Sign of Bronchial Obstruction.

I regard loss of vocal fremitus over an area still resonant, or even tympanitic, as one of the earliest and best signs of bronchial obstruction—a wrinkle I got many years ago from my old teacher and friend Dr. Glynn.

("Mediastinal Tumours," by John Hill Abram, M.D.Lond., F.R.C.P., 'Clin. Journ.,' vol. xxxv, p. 401.)

(154)

The Wisdom of Doubt.

It will be well for us to remember the words of Pasteur: "In experimental science it is always a mistake not to doubt when facts do not compel you to affirm."

("The Geographical Distribution and Ætiology of Cancer," by F. M. Sandwith, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxv, p. 409.)

(155)

The Tendency to revert to the Normal.

Speaking generally there is undoubtedly a tendency in the individual who has some morbid variation from the normal to reproduce that variation in his offspring, although Nature is for ever trying to revert to the normal, and in favourable environment she succeeds in the generality of cases in restoring the normal condition. Hence you see, even in families where a morbid tendency is strongest, most of the members usually escape.

(*Ibid.*)

(156)

Heredity and Cancer.

Dr. Lazarus Barlow lately gave an interesting account of the work done in the Cancer Research Laboratory of the Middlesex Hospital, and, referring to the investigations upon heredity in cancer, he said that the examination of the records of 8000 cancer cases of which the Hospital had notes showed that there was no evidence that the disease was inherited. From the point of view of life assurance and of allaying a general fear, this statement is of great public utility.

(*Ibid.*)

(157)

Cancer and Arboreal Tumours.

The connection between the prevalence of cancer and the prevalence of arboreal tumours has been noted by many observers. The vegetable tumours are apparently contagious, as several usually exist in neighbouring trees, and it is supposed that the infection is borne from tree to tree by insects.

(*Ibid.*)

(158)

Beware of the dangers of Eclecticism.

I need hardly warn you against being misled by those who choose particular instances to prove their arguments. If you are allowed to choose particular instances you can prove anything. To prove cases by the choice of particular instances the audience must be ignorant and stupid, which you are not. The instances which I am about to give are mere illustrations. You may have heard the story of the philosopher who was taken to see the temple of Neptune. Around the walls were the pictures of those who had made dangerous voyages in ships, like those of St. Paul, and naturally they were glad whenever they touched land again, and their votive offerings were hung in the temple of Neptune. The philosopher was asked to gaze on the pictures and acknowledge the goodness of the sea-god, the preserver of the givers. But he said, "Now show me the pictures of those who went to sea and did not come back?" So when wonderful cures are told, ask about those who were not cured.

("Some Fundamental Truths in Surgery," by C. B. Lockwood, F.R.C.S., 'Clin. Journ.,' vol. xxxv, p. 1.)

(159)

The Ideal Lunatic Asylum.

Talking of lunatics who are operated upon, Mr. Lockwood says: "You see occasionally advocated—I saw it recently advocated—that the State should at once deal with the mentally afflicted and get rid of them. I suppose the ideal lunatic asylum of that person would consist of a receiving room, a lethal chamber, and a crematorium. But just imagine, if once you began to condemn human beings to death because of their mental condition, where would you end? Surely I cannot help suspecting you would find people who might refrain from operating because they did not like the person's nationality, or perhaps his theology, or his political creed. Surely we, as medical men, have nothing to do with these things. It is our bounden duty, when brought into contact with a dying person, to try, if possible, to save his life, quite irrespective of his mental condition."

(*Ibid.*)

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CHRONIC INTESTINAL STASIS.*

By W. ARBUTHNOT LANE, M.S.Lond., F.R.C.S.Eng.,
Surgeon to Guy's Hospital.

I EMPLOY the term "chronic intestinal stasis" to indicate such an abnormal delay in the passage of the intestinal contents through a portion or portions of the gastro-intestinal tract as results in the absorption into the circulation of a greater quantity of toxic or poisonous materials than can be treated effectually by the organs whose function it is to convert them into products as innocuous as possible to the tissues of the body.

When these poisons and their products of conversion exist in excess in the circulation they produce degenerative changes in every tissue and in every organ in the body. It is probable that the textures of those organs whose business it is to convert, carry, and eliminate them suffer more than do the other tissues of the body which are merely permeated by them. In proportion as the stasis is prolonged, so under the influence of a progressive strain greater than they are able to bear these several organs undergo a degeneration which proceeds with increasing rapidity as the condition advances. What the organs are which convert and excrete these poisons, what share each takes in the process, and how any organ is affected in its physiology, is very difficult to define accurately, especially as far as the ductless glands are concerned. We believe that the liver is the most important converter of the poisons, and that the kidneys and skin are the chief excretors of the products of conversion.

As to the part taken by the thyroid gland, the pituitary body and the suprarenals we possess little precise information.

Our observations in this direction are purely experimental, and if our several operations on the

* Delivered before the Medical Society of London.

drainage scheme have done nothing more than demonstrate the damage done by these poisons, the remarkable power of repair which the several tissues of the body exhibit on being freed from their malign influence, and the extraordinary improvement in the functioning of the organs of the body after operation, they have been fully justified. They have, however, done more than this, since they have thrown a light upon intestinal conditions which has simplified their treatment most materially, and has put them upon a definite mechanical basis, replacing what was previously little more than a nomenclature.

As corroborating the wisdom of attending to the drainage scheme of our bodies I would call attention to some of Carrel's experiments, since they throw a most important and instructive light on the subject. When I saw Carrel, more than two years ago, he showed me living tissues growing in a sterile medium on a microscopical slide. At that date they had been growing for twelve days. I asked him what would result from the defective drainage of the tissue, since the quantity of serum was limited and the products of growth must be ejected into it. He said he did not know, but expected to learn shortly. After a few days he found that the vitality of the tissues became diminished, and that the growth of the cells was slowed, and that it ceased very soon after. Death of the tissue took place obviously because of the imperfect drainage, just as our bodies and tissues become depreciated and finally die from chronic intestinal stasis. To meet this, when growth commenced to flag he opened up the specimens of growing tissue and washed away the toxic products. By so doing he gave the material another period of growth extending over about fourteen days. This he was able to repeat many times till death finally took place. A line indicating the extent of growth was for a time level, then descended gradually and finally very rapidly, illustrating exactly what takes place in chronic intestinal stasis in the human. By attending more carefully to the drainage of the toxic products of his specimens he was able to carry the growth along a horizontal line with regular undulations. Again, by feeding them with the juice of fœtal thyroid or spleen, he increased the growth of these tissues in an extraordinary manner, the increase during twenty-four hours varying between three and forty times the bulk of the original material. In this way he replaced the gradually descending line of tissue-growth ending in death by one which was rapidly ascending and showed no indication whatever of terminating in death of the tissue. In other words, by attending carefully to the drainage and by giving suitable foods, he gave that tissue a perpetual life and a rate of growth enormously in excess of the normal. I am relating this to show how very simple and yet how very important is the problem involved in the consideration of chronic intestinal stasis. I have just seen him perform this experiment on a larger scale when he eviscerated a cat, and so arranged the drainage of the intestine that it continued to perform its function normally during artificial respiration for a period of twelve hours. He hopes by preventing the death of the organs by peritonitis to prolong their life for a much longer period. The improvement in the growth of tissue which he obtains by attending very carefully to the drainage is well illustrated in the very rapid and great increase in the growth and weight of the child suffering from tubercle or rheumatoid arthritis which takes place after the large bowel has been excluded from the gastro-intestinal tract.

I found the most satisfactory confirmation of my views in the study of Carrel's magnificent work. It also suggested many points of great interest in the factors affecting the growth of malignant disease, since several of his growing tissues were sarcomatous. He will supply much valuable information on this subject very shortly.

I will now revert to the study of several important points in intestinal stasis in the human subject. Personally I believe that unless the capacity of the several tissues of the body to resist the entry of certain organisms is inhibited by the auto-intoxication resulting from intestinal stasis it is impossible for these diseases to develop. I refer more particularly to tubercle, rheumatoid arthritis and similar diseases. Therefore to meet these diseases I adopt means to improve the drainage scheme, whether simply mechanical or operative, with the most excellent results. This is nowhere better exemplified than in the extraordinarily rapid disappearance of large tubercular glandular masses in the abdomen after disconnection of the big bowel. The disease disappears and the health and weight of the patient improves correspondingly, and if the subject be a growing child the rate of growth is

much greater than before. This matter has been already fully dealt with by myself and others. Certain symptoms of auto-intoxication present problems of great interest as regards their causation.

Pigmentation of the skin becomes a very marked feature in advanced cases of stasis, especially in patients with dark hair. These cases have occasionally been diagnosed as Addison's disease by very competent physicians, and I have no doubt would have gone to the post-mortem room as such except for operative interference. On eliminating the supply of poison the colour of the skin changes with a remarkable rapidity. The deep brown or coppery tint disappears and is replaced by the warm red colour indicative of health. Has this unmistakable change come about by a restoration of the suprarenal to its normal condition, or to what is it due?

Following almost immediately on the exclusion of the large bowel, the cold blue clammy hand is replaced by a nice warm pinkish dry one. Nothing can be more obvious than this change. Is this remarkably rapid alteration due to an improved cardiac condition alone, or does the thyroid, adrenal or the pituitary body exert any influence upon it? It is not a matter of food, as the patient may be having nothing but normal saline. In the case of a young man with fingers that had been blue for some years, on asking him what was the first change he noticed within a day of the operation, he said, "My blue fingers have gone," and the blueness never recurred.

We know very little of the action of the ductless glands except from physiological experiment, and perhaps nothing in the operative treatment of chronic intestinal stasis strikes one as more disappointing than the helplessness of the physiologists to supply us with useful information that can be applied to our practice.

Again, in the case of the kidney affected by so-called Bright's disease, which is merely a product of chronic intestinal stasis, the exclusion of the large bowel is followed by an improvement in functioning which is as extraordinary as it is rapid, and a patient who has been face to face with death is quickly restored more or less completely to health, usefulness and activity.

The great difficulty in the treatment of chronic intestinal stasis and its results is to recognise when it is too late to interfere; in other words, when the end result has assumed such proportions that the removal of the primary cause does little or no good. As an extreme instance, take cancer of the breast, or of the ducts of the liver or of the pancreas, all products of chronic intestinal stasis in the first instance. What would be gained by eliminating the absorption of toxic material at this stage? In the antecedent phases, however, the hopeless conditions which form the last chapter in the story of chronic intestinal stasis may be readily avoided. Take the breast while it is indurated and nobbly as it is in marked chronic intestinal stasis, eliminate the supply of toxins, and a soft healthy organ results which need cause the patient no anxiety in the future. As far as I know, a healthy breast in a subject showing no evidence of stasis does not become cancerous.

Again, as regards the influence of these toxins or poisons on the nervous system, I have seen a patient who had been confined to bed for many months having neither the capacity nor desire to stand or walk, and whose mental condition was such that she was regarded by many as an imbecile, become a happy, active, intelligent woman within a few weeks of the removal of the large bowel. Since that operation she has been leading a useful life and earning her living.

A woman was sent to me from South Africa in order that I might remove the Gasserian ganglion for epileptiform neuralgia of the right fifth nerve. She had suffered constant pain with exacerbations of great severity for about nine years. During the last two years her condition had become intolerable. She was definitely static, and her nervous symptoms varied with her toxicity. She was short-circuited. After the operation she had pain in the face, slowly diminishing for a week. On the eighth day it disappeared. She had no exacerbation during that week. She made a remarkable recovery interrupted only by a short slight attack at the end of the fifth week. Her general health and weight have improved rapidly, and a happy smiling face has replaced an expression of hopeless misery.

In another patient, a man, headaches were intense and associated with vomiting. One of the most distinguished of our nerve physicians considered them to be due to a tumour in the frontal lobe and advised operation, which was, however, refused. The intense pain in the head disappeared abruptly after an ileo-colostomy.

These cases are caricatures or extreme conditions, but it is from the extreme conditions one learns most. The same or similar symptoms exist in a lesser degree in most advanced cases of stasis.

In the case of the thyroid I have seen a large adenoma of this organ subside with great rapidity after removal of the large bowel, and typical symptoms of exophthalmic goitre of long standing associated with intestinal stasis have also rapidly and permanently disappeared in other cases.

Such, then, are the results of absorption from the alimentary tract. What is the source from which these poisonous products are obtained? They may be absorbed from any portion of the gastro-intestinal tract as a primary absorption from the intestine. This supply may be supplemented by absorption of poisonous products from the secondary results of their absorption, such as from suppurating gums or from any secondary infective process communicating or not with the gastro-intestinal tract.

The absorption from the secondary result of auto-intoxication may in some cases be very considerable, and the removal of the absorption from the secondary focus may result in great improvement in the patient's condition, though the source of absorption from the intestine be uninfluenced by its treatment. The primary absorption from the intestine varies in importance, not with the degree of stasis, but rather with the unfitness of that portion of the bowel to deal with organisms, etc., to which it is unaccustomed. For instance, solid material may remain in the sigmoid flexure or pelvic colon for a considerable time and produce but a moderate amount of auto-intoxication. On the other hand, the infection of the contents consequent on a very moderate stasis in the small intestine may bring about a very severe condition of auto-intoxication while at the same time the mechanical symptoms resulting from it are also much more conspicuous.

I wish particularly to call your attention to the situation of the sites of the sources of excessive absorption of toxins from the gastro-intestinal tract. Since the profession has begun to study the subject of auto-intoxication in chronic intestinal stasis it has been inclined to assume that the large bowel is the sole source from which these toxins are derived, and it has got into the habit of looking on stasis as merely colonic. In consequence of this operations have been conceived chiefly with the object of facilitating the effluent in the colon, and in this manner the chief site of absorption has been overlooked and left untreated. This is largely due to Metchnikoff's views on the subject. He considers that if we had no large bowel we would live longer. This is perfectly true, as evidenced by the fact that the removal of the large bowel results in a marvellous improvement in the health and appearance of the individual and in the duration of life. But I would point out that the benefit which results from the removal of the large bowel does not show that the colon is the chief source from which toxins are absorbed in excess. Indeed in a considerable proportion of cases I believe the bulk of the absorption takes place from the small intestine, and that the control of the effluent in the ileum is the fatal product of civilisation.

Stasis of the small intestine with the associated infection of its contents by organisms to which it is unaccustomed is not primary, but is secondary to a stasis in the large bowel. In other words, if it were not for the presence of the large bowel the conditions producing stasis in the small intestine would not arise. If the cæcum did not become overloaded the obstruction to the ileal effluent, either by an acquired mesentery, an appendix hitching it up, or by simple stasis, would not develop. Consequently the contents of the small intestine would not become infected by organisms, the duodenum would not be blocked by the drag of the small intestines obstructed at the end of the ileum, the mucous membrane of the duodenum would not inflame and ulcerate, the biliary and pancreatic ducts would not be infected, and the obstructed outflow from the stomach with all its associated sequelæ would not occur.

Now, I wish to show that the extraordinary improvement that results from short-circuiting and the disconnection or removal of the large bowel is due largely to the fact that the evacuation of the small intestine is facilitated by its introduction into the pelvic colon, and that the infection of its contents by organisms which grow in the stagnating material in the large intestine ceases abruptly. I do not wish to suggest that all absorption of toxins takes place from the stomach and small intestine, but I do maintain that the tract other than the colon plays a very important part, and I believe the more important part in the process of absorption. It appears to me that the point of greatest

difficulty in the passage of material along the gastro-intestinal tract is through the last few inches of the ileum. This is particularly the case when the cæcum has been securely fixed by acquired adhesions in the iliac fossa. In such cases the delay of the effluent at the pelvic brim may be very great; indeed, in one of my cases which Dr. Jordan has examined with bismuth and the X rays, the material remained in the terminal coil of the ileum for as long as eighty-five hours * without there being found at the operation any evidence of interference with the effluent by an acquired peritoneal band, or by an appendix fixed in such a position behind the small bowel as to control the passage of material through it in certain positions. Now, this form of simple obstruction, which cannot be recognised at the time of the operation and can only be determined by bismuth and X rays, I call the "simple static variety." I use this term as opposed to the more obvious variety, in which the bowel is controlled by an acquired band or by an appendix, either of which is readily recognised when the abdomen is opened, though the extent of its effect on the effluent can only be gauged by bismuth and X rays.

Since with an apparently uncontrolled ileal termination the delay in the effluent may be very considerable, one can readily see how a structure which by strain or pressure exerts apparently but a comparatively slight constricting influence on the lumen of the bowel may affect very materially the passage of the contents through it. The duodenum ends usually in such a manner that its effluent can be controlled by the exercise of a vertical downward traction upon the jejunum. I believe that to obviate this mechanical disability the commencement of the jejunum becomes attached progressively to the posterior abdominal wall by an evolutionary process, so that, in the highest state of development, having become attached to the under surface of the transverse mesocolon in a direction from left to right, this portion of bowel is fixed in the form of a semicircle.

I have indicated diagrammatically what I believe to be the normal condition at birth; and the condition commonly present in advanced duodenal obstruction in chronic intestinal stasis. The arrows indicate the acquired ligament, which represents the crystallisation of lines of force, which is frequently present and which is evolved to take strain off the termination of the fixed bowel. The result may be fully developed fixation of the jejunum to the under surface of the transverse mesocolon. The acquired mesenteric bands which are evolved to fix the bowel in this situation obviate angulation at its termination and consequent interference with the effluent. The advantageous mechanical arrangement afforded by this last type may be imitated by suturing the jejunum to the under surface of the transverse mesocolon after the division of any such bands, should they exist. It would seem impossible to eradicate from some minds the idea that these acquired bands and mesenteries are inflammatory or congenital in origin and not evolutionary during the lifetime of the individual.

I do not propose to discuss here the details of the several operative procedures which I employ, as I have done so on very many occasions.

We may differ widely in our opinions as to the causation of the several conditions which produce mechanical changes in the intestine, as to the mode of absorption of poisonous products, and as to the best modes of treating the several troubles as they arise, but I trust that the discussion, which I feel sure will be a very full one, will help us to materialise our views on a subject which I believe to be one of the biggest and most important which we have handled recently. I have just returned from the United States, where chronic intestinal stasis is receiving the closest attention, and where it is being dealt with in a very practical manner by the most progressive surgeons in the world.

Mr. H. G. PLIMMER spoke of his experience of the post-mortem appearances of the alimentary tract of animals. In mammals, as the result of about 150 post-mortem examinations, he had found that constipation was rarer than in post-mortem examinations on the human subject, but enteritis was commoner. Of about 250 primates ninety-two died from enteritis, the colon being the chief seat of the affection. In animals there was a greater percentage of actual organic obstruction — *e.g.* intussusception. Rickets was common among the younger animals, and was associated with constipation of both large and small intestine, more especi-

* "Radiography in Intestinal Stasis," by A. C. Jordan, M.D., 'Proceedings of the Royal Society of Medicine,' 1911, vol. v, p. 20, fig. 14.

ally of the large intestine. The only symptom of prolonged constipation in them was severe anæmia. In nearly all the cases of enteritis suppuration of the gums was present, to which he attributed the enteritis. In his experience he could not trace a closer relationship between cancer and obstruction or constipation than between it and other conditions.

Dr. ALFRED C. JORDAN said that the most useful contribution he could offer to the discussion was to show on the lantern X-ray photographs obtained by himself in the course of his investigations of the gastro-intestinal tract in cases of intestinal stasis. He entered upon this branch of work without the vaguest idea whither it would lead him; the results began to present themselves before him automatically, and he was quite unable to interpret them until he had followed some of the cases to the operating theatre and seen the actual state of the parts. He then found that Mr. Lane was able to show him many abnormalities which he had failed to detect by X-ray examination, and the task he had to face was that of developing the method of examination so as to show all parts of the gastro-intestinal tract in turn, and to discover all abnormalities of structure and function. At the present stage no fact had become so firmly borne in upon him as that of the interdependence of the several parts of the gastro-intestinal tract. The time had passed by when an examination, say of the stomach alone, could be considered satisfactory; the different parts reacted upon one another, and abnormalities of the intestines were an essential factor in the production of disorders of the stomach. He had been most particularly struck with the relation which was found to exist between the duodenum and the lower end of the ileum. Mr. Lane had already explained the nature of the relationship; he (Dr. Jordan) would hope to show actual photographs illustrating it in an irrefutable way. Briefly, his experience showed that the duodenum was the most sensitive part of the intestinal tract; whenever stasis occurred in the ileum, with consequent overloading of the lower coils of the ileum, the downward pull upon the mesentery of the small intestine caused the jejunum to be pulled vertically down at its commencement, and a sharp kink was produced at the duodeno-jejunal junction with distension of the duodenum. In severe cases he found the duodenum contracting actively in its endeavours to overcome the effects of this kinking. In the most severe cases the duodenal contractions amounted to strong writhing movements. He had observed the duodenum writhing for nine hours, with great pain to the patient, and after the bismuth had finally succeeded in passing through the duodenum, he still observed the duodenum writhing, although it contained nothing more opaque than Benger's food. This was on the day following that on which the bismuth had been administered, when it was contained in the lower coils of the ileum, with only a moderate quantity in the large intestine. The reaction of the duodenum to stasis in the lower coils of the ileum was so constant that it formed a trustworthy indicator, equally valuable whether positive or negative. That is to say, the discovery of a normal duodenum was a sign that intestinal stasis was not likely to be present, while the observation of a distended (or "static") duodenum was a certain indication that other evidence of intestinal stasis would be found at the subsequent examinations, especially at the lower end of the ileum, although one could not predict, in any individual case, the exact cause of the ileal stasis to be revealed at these subsequent examinations. The three forms of ileal stasis had already been described by Mr. Lane. Simple ileal stasis was a factor in all cases. The occurrence of an ileal kink was an additional disability of great importance to the patient. In its early stages an ileal kink was only operative in causing obstruction when the patient was upright. In the cases in which the appendix wound round the end of the ileum in such a way that it obstructed the flow from the ileum into the cæcum, the obstruction was far greater when the patient was upright: in well-marked cases the terminal coil of the ileum was greatly hypertrophied, and was marked by a groove where the appendix had pressed upon it. Duodenal distension might be present without causing any subjective symptoms; ultimately, however, congestion occurred in the distended mucous membrane. The duodenal contractions were then painful, and when the congestion had gone on to the stage of ulceration the pain was usually a marked feature. The last stage was that of cicatrisation, and he had several instances to show of ileal stasis (by a kink or by the appendix) in the subjects of stenosis by a cicatrised duodenal ulcer. The general effects of intestinal stasis had been described by Mr. Lane.

He (Dr. Jordan) was accustomed to find radiographic evidence of atheroma of the aortic arch in those subjects of chronic intestinal stasis who were past middle age. This was an important fact, for patients with aortic dilatation were bad subjects for operation. With regard to treatment he had nothing to say except to refer to the action of liquid paraffin. In persons who were taking this substance (or had done so within two or three weeks) it was often impossible to obtain radiographic evidence of intestinal stasis, although the patients might present the typical features of the disease. Mr. Lane recently operated on a man in whom he (Dr. Jordan) had found no abnormality of the duodenum or ileum, but the patient was found to have a dilated duodenum and a well-marked ileal kink. The administration of liquid paraffin had been so efficacious in this case that it had abolished the stasis for the time being. In advanced cases the curative effect of this treatment was less complete. The beneficial effect of rest in bed was frequently seen on X ray investigation in cases of duodenal distension. In the case of out-patients the characteristic "writhing" duodenum was often seen, and there was the greatest difficulty in the passage of the bismuth emulsion through the duodenum. The stomach felt the same difficulty, and was found to be hypertrophied from its undue efforts to force its contents into the over-filled, distended duodenum. After such a patient had lain in bed for three weeks the duodeno-jejunal kink was found to have passed off (except in the worst cases), and the bismuth was seen to pass through the duodenum and into the jejunum without difficulty, although the large size of the duodenum might be apparent still. The gastric hypertrophy did not pass off so soon, however; the stomach still contracted more vigorously than normal; consequently the stomach then emptied itself through the duodenum into the jejunum more quickly than normal.

(A series of lantern-slides was then shown illustrating the relation between the duodenum and the ileum in intestinal stasis, with examples of the distended, the congested, the ulcerated, and the cicatrised duodenum, followed in each instance by the picture showing the ileal stasis—either simple ileal stasis, or stasis with an ileal kink, or stasis produced by the appendix. The effect of the upright posture in causing obstruction in the presence of an ileal kink was also illustrated.)

Mr. H. W. Carson said he had no doubt that great advances had been made since the use of bismuth and X rays in the investigation of these cases. It was a question whether stasis could occur in the absence of a developmental fault, and also whether the bands causing kinks were inflammatory or non-inflammatory in origin. He believed with Mr. Lane that infection played no part in the formation of these adhesions. The youngest patient in whom he had been able to make out the presence of a kink was twenty-two, the average age being between twenty-five and thirty-five. It was difficult to diagnose between an ileal kink and appendicitis. In cases in which an operation was done, believing chronic appendicitis to be present, a thorough exposure of the parts by free incision was necessary. An important point was the real dependence between the upper and lower part of the alimentary tract. If the ileal kink was due to dropping of the cæcum, which he believed, it was not enough to divide the cause of the kink, but the cæcum should be fixed also.

Mr. H. H. Sampson spoke of the relation of intestinal stasis to *B. coli* infection of the urinary tract. He said he proposed to confine his remarks to the discussion of the relation between intestinal stasis and *Bacillus coli* infection of the urinary tract. In the consideration of the ætiology of this condition the bowel suggested itself at once as the most likely source of the infection. This was widely recognised in the treatment of bacilluria, regulation of the bowels being an important factor in the amelioration of the condition. At St. Peter's Hospital, where a large number of cases of bacilluria were seen in the out-patient department, he had noticed that the majority presented some signs of intestinal stasis. When questioned as to the condition of their bowels many had volunteered the statement that the subacute exacerbations, which were such a marked feature of the disease, generally occurred when the bowels were constipated. One patient insisted that he had no attack so long as he secured a free daily evacuation of the bowels. Jeffreys, in the '*Quarterly Journal of Medicine*,' April, 1911, reported 60 cases of coliform infection of the urinary tract in children. Of these, 34 showed evidence of bowel trouble. In John Thomson's 33 cases, reported in 1902 and

1909, 19 suffered from habitual constipation. Jeffreys, describing the general condition of the children in his series of cases, said they were "generally wasted, pale, or with an earthy tint of the skin." In view of these facts it occurred to Mr. Sampson that a thorough investigation of some cases of bacilluria might throw clearer light on the relation between intestinal stasis and *Bacillus coli* infection of the urinary tract. Bacilluria was a very common condition in children. In them it gave rise to rather obscure symptoms, such as abdominal discomfort, which were apt to be misconstrued unless bacilluria was kept in view as a possible cause. It was in children that he had had the opportunity of carrying out a fuller investigation of the intestinal condition present in cases of bacilluria. His first two cases were good examples of persistent intractable *coli communis* bacilluria which resisted prolonged and energetic treatment. After the exclusion of other urinary diseases by radiography and cystoscopy, treatment commenced in each case with daily irrigation of the bladder, followed by the instillation of iodoform emulsion. The bacilluria persisted or returned after a short interval. In the one case Mr. G. E. Waugh then removed the appendix with the idea of cutting off an obvious source of infection. In the other case also, acting on Mr. Waugh's advice, the appendix was removed. He understood that Mr. Waugh had had a large proportion of permanent cures from this procedure, but in both these cases the bacilluria persisted. In each case the complexion of the child was fair, with no apparent increase in cutaneous pigmentation. Both children were wasted and showed some degree of anæmia. There was marked coldness of the hands and feet, with general listlessness and mental apathy. He showed radiograms of these two cases taken after a bismuth meal. In the one case it was noted at the operation that the ascending colon was provided with a long loose mesentery, and five hours after the meal there was bismuth in the cæcum which was lying in the pelvis. Nine hours after the meal the cæcum appeared to be displaced still more to the left side, and some bismuth was seen in the terminal portion of the ileum. There was also bismuth in the transverse colon and splenic flexure. Thirteen hours after the meal the food was seen to be crowded in the colon between the hepatic flexure and the iliac colon, suggesting some mechanical difficulty in the left iliac fossa. Twenty-nine hours after the meal the bismuth had moved on a little and extended from the transverse colon to what he took to be a loop of the pelvic colon. Fifty-three hours after the meal the bismuth extended from the descending colon to the pelvic colon. Seventy-seven hours after the meal the bismuth occupied the pelvic colon and rectum, and it had all been passed at the end of 101 hours. He thought it could reasonably be concluded that in this case there was marked stasis in the large intestine and a delay of at least nine hours in the lower end of the ileum. In the second case a bismuth meal again revealed stasis in the large intestine. Mr. Waugh kindly allowed him to cystoscope this patient. He found that the right ureteric orifice was swollen and hyperæmic, and that the left orifice was normal in appearance. He catheterised the right ureter, and a pure culture of *B. coli communis* was obtained from the urine of the right kidney. He thought this was of interest, because there appeared to be some obstruction at the hepatic flexure. In a radiogram taken twenty-nine hours after the meal no bismuth had passed beyond the ascending colon. This might be coincidence, but certainly suggested the possibility of local lymphatic infection from colon to right kidney. The food passed rapidly from hepatic flexure to lower pelvic colon and rectum, but still remained in the rectum seventy-seven hours after the meal. The next two cases were brought to hospital for the relief of nocturnal enuresis of several years' duration. The usual remedies were tried without relief. Bacteriological examination of the urine revealed in each case *B. coli communis* in pure culture. No other urinary disease could be discovered. Both these children had dark brown hair, and showed marked increase in cutaneous pigmentation, especially on the forehead and neck. The hands and feet were cold and clammy. The children often complained of feeling cold, and one suffered from chilblains every winter. In one case a bismuth meal was given and showed definite ileal delay for at least nine hours. This was a good illustration of Lane's ileal kink, and showed the terminal portion of the ileum rising out of the pelvis to its oblique junction with the cæcum. There was no marked delay in the large intestine. The other patient was subject to bilious attacks, was prone to feel ill when she travelled in any vehicle,

and occasionally complained of pain in the left side of the abdomen. In this case the urine from the left kidney contained *B. coli communis*, while that from the right kidney proved to be sterile. A bismuth meal revealed an ileal delay of from nine to thirteen hours. There was no marked delay in the large intestine. The fifth case, a girl, æt. $3\frac{1}{2}$ years, was admitted to hospital with a subsiding pulmonary condition. Pus was found in the urine, and bacteriological examination disclosed a pure culture of *B. coli communis*. She was being treated with an autogenous vaccine, but up to the present showed no signs of improvement. This patient presented marked increase in cutaneous pigmentation all over the body. She had cold hands and feet, with a sudden transition from warm to cold on each arm at the level of the lower border of the deltoid. She showed marked lassitude and mental apathy. A bismuth meal disclosed marked delay in the large intestine, the bismuth not being evacuated for from seventy-six to one hundred hours. The sixth case was admitted with a history of several attacks of renal colic. Examination of the urine showed the presence of a coliform bacillus, not *B. coli communis*. Cystoscopy and radiography disclosed nothing abnormal. The child showed no signs of intestinal stasis. A bismuth meal indicated no delay in the alimentary canal. Intestinal stasis, therefore, was definitely present in five cases of bacilluria due to the *B. coli communis*. There was no stasis in one case of coliform bacilluria. He did not suggest that intestinal stasis would be found in every case of *coli communis* bacilluria. His remarks were intended to apply to the chronic intractable bacilluria with periodic subacute exacerbations, where the underlying pathological condition was probably a unilateral or bilateral pyelitis. In these cases he did suggest that intestinal stasis was not only the primary cause, but also the essential factor perpetuating the condition.

Dr. R. MURRAY LESLIE said that the subject of intestinal stasis was extremely important from the standpoint of the physician. The brilliant researches of Mr. Arbuthnot Lane had thrown new light on the whole question of functional disturbance of the gastro-intestinal tract, of which dyspepsia and constipation were merely symptomatic manifestations. Intestinal stasis consisted essentially in the arrest of normal peristalsis or such interference as to render the peristaltic movements inefficient in preventing undue accumulation of intestinal contents. The stasis might be produced either by neuro-muscular atony or by mechanical obstruction. Neuro-muscular atony might be congenital or it might be associated with anæmia and other debilitating conditions, or, indeed, might be the result of sedentary habits, especially when associated with matutinal neglect. The nervous factor might manifest itself by a defective reaction to natural stimuli, which might be either natural to the individual or due to a temporary lowered vitality of the nerve centres, as in neurasthenia. Dietetic errors, including the taking of semi-solid pulpy food containing an insufficiency of solid stimulating residue, also played an important part. The condition known as enterospasm, which in women might take the form of spasmodic constriction of a portion of the colon, was a not uncommon accompaniment of hysteria and neurasthenia, particularly when these were associated with painful pelvic affections; and if, as sometimes happened, atony of one part of the large bowel accompanied enterospasm of the portion immediately beyond, there was present a combination which specially favoured the occurrence of intestinal stasis. As regards mechanical obstruction, it was only within the last few years that the important *rôle* played by mechanical obstruction and its far-reaching effects had begun to be realised. In common with many others, he was at first extremely sceptical as to the presence and significance of the various adhesions and kinks described by Mr. Lane, but six years ago he became a convert as the result of his observation of a particular case of chronic intestinal stasis of twelve years' standing. All medical and hygienic remedies having proved unavailing, he consulted Mr. Lane, as the patient appeared to be sinking from emaciation and asthenia. At the short-circuiting operation several thick adhesive bands were clearly demonstrated, which were certainly abnormal. The patient, who had been a miserable chronic invalid for many years, now enjoyed perfect health, and had been able to play seven sets of tennis without undue fatigue. She gained 2 st. in weight after the operation. His later experience of a considerable number of similar operation demonstrations, in most cases preceded by radiographic examinations, had only tended to strengthen his belief in the importance of these adhesive structures and kinks, and he felt

he could with confidence predict that a similar view would sooner or later be universally accepted by the medical profession. In his own experience the two facts that stood out prominently were the enormous preponderance of cases occurring in the female sex and the very frequent association with enteroptosis. Out of some fourteen successive cases of well-marked intestinal stasis that had recently been under his care all had been women, and with the exception of three all exhibited X-ray evidence of visceroptosis to a greater or less degree. The average age of these patients was thirty-five, the ages varying from twenty-nine to forty-four. He had one case (not in this series) of an old lady, æt. 72 years. In the above series of cases the average period between the onset of symptoms and the date of coming under observation had been twelve years. There had generally been a history of constipation, commencing in early adult life (eighteen to twenty-four), the more remote symptoms of auto-intoxication, including emaciation and asthenia, becoming pronounced between the ages of thirty and forty-five. Another curious fact in his series was that with three exceptions all the patients were unmarried, which confirms Mr. Lane's views in regard to the beneficial effects of matrimony as a counteracting influence. The explanation of this sex preponderance he believed to be found in the fact that enteroptosis was probably by far the most important primary causal factor in the production of intestinal stasis, visceroptosis being fully ten times more common in the female than in the male. This greater frequency might be attributed to the following causes: (1) The less marked obliquity of the female pelvis, so that the pelvic cavity, in addition to having greater width and capacity, was more of a continuation of the elongated female abdominal cavity, while the more expanded pubic arch gave less direct support to the overlying viscera. (2) The more lax condition of the abdominal walls in women, which offered less resistance to downward pressure, and which might be accentuated by over-distension due to repeated pregnancies and other causes. (3) The wearing by women of wrongly constructed corsets, which Einhorn believed to be an important causal factor, particularly when the corset exerted pressure on the base of the thorax and upper part of the abdomen, thus forcing the organs downwards. (4) The almost universal adoption in civilised countries of the sitting posture in the act of defæcation in contrast to the natural crouching one, the present high lavatory seats being specially harmful in the case of women and children. And (5) the greater tendency to neurasthenic debility and constitutional weakness of the mesenteric and other peritoneal attachments. As regards the symptomatology of intestinal stasis, his series of cases tended to confirm the truth of Mr. Lane's observations in regard to auto-intoxication, though he was inclined to join issue with him in regarding the frequently associated neurasthenia as necessarily in all cases a sequela of intestinal stasis. He believed that in not a few instances neurasthenic debility was in reality a precursor of the stasis and a not unimportant casual factor—possibly hereditary. It would be impossible to enumerate all the symptoms manifested by these patients; the most important had been general languor and inertia, irritability, mental depression, cold, clammy and livid extremities, dark staining of the skin, muscular debility, incapacity for prolonged physical or mental exertion, and progressive emaciation. A nodular condition of the breasts was often present in the advanced cases. Abdominal pain and tenderness were present in varying degree, and in practically all of these was found evidence of definite obstruction, due to ileal kinks, duodeno-jejunal kinks, angulation of the flexures, or fixation and impaction of the cæcum or sigmoid in the pelvis. In several cases the radiographic evidence was confirmed at the subsequent operation. It was noteworthy that in some cases of enteroptosis and intestinal stasis there were remarkably few symptoms, while other patients, in some of whom the condition seemed less advanced, were in a miserable condition of ill-health; in the former a condition of tolerance seemed to have been established. He might take this opportunity of emphasing the great importance of radiography in diagnosing the presence of visceroptosis, the presence of kinks or impactions, the site of the obstruction, and the full extent of the stasis. As regards remote sequelæ, he had had instances of cystic changes in the left ovary which had become involved in the sigmoid adhesions, appendicular irritation, gall-stones, gastric dilatation, and mucous colitis, but had had no experience of actual ulceration of stomach or duodenum, carcinoma, or tuberculosis following intestinal stasis. With reference to treatment, he could speak

strongly of the value of liquid petroleum in association with abdominal massage, exercises and electricity, while in the visceroptosis much benefit resulted from the use of properly constructed corsets and abdominal supports. As regards operative measures, resection of the bowel or even short-circuiting was heroic treatment even for obstinate constipation. He had, however, had experience of several cases in which simple measures had been tried without avail to relieve the constipation and abdominal pain and to check the progressive ill-health, and in which he felt it his duty to advise operation. The dividing of the ileum and the short-circuiting of its termination into the pelvic colon or upper part of the rectum had proved more than satisfactory, and the patients were all now enjoying good health. In Mr. Lane's hands the operation appeared to be attended by but little risk. If the results were so good it was surely better in advanced cases where simple remedies had failed to have recourse to this radical cure for intestinal stasis, rather than leave the patient in a miserable state of suffering and chronic invalidism, rendering life almost insupportable, and so lowering the resistive power of the tissues, owing to the effects of auto-intoxication, as to frequently endanger the life of the individual from intercurrent complications or otherwise.

Dr. F. J. SMITH said: I think the Medical Society of London is to be congratulated on having been the recipient of Mr. Lane's paper, and while I criticise it I must not be taken to be unappreciative of the careful thought which he has brought to bear on the problem of intestinal stasis, nor to be wanting in admiration for the boldness, skill and thoroughness with which he has applied his surgical talents to the relief of the condition; but all the same I must confess to having doubts in my own mind about the fundamental canons of Mr. Lane's position. In the first place, I can hardly bring myself into the position of admitting that cancer of the breast is directly caused by intestinal stasis in any degree, though one may be ready to admit that the general metabolism of the body is improved by a free passage along the intestine. Again, I have seen far too many cases of pyorrhœa and the beneficial results accruing from the cure of the condition to accept this as secondary to intestinal stasis. In my opinion, pyorrhœa is distinctly and definitely a condition totally independent of constipation, and yet responsible as *the* causative factor for a large number of diseases for which Mr. Lane would resort to colectomy. I have also found pyorrhœa definitely and distinctly curable by measures falling very far short of an abdominal section with or without the release of adhesions or removal of the colon. I agree with Mr. Lane that Bright's disease and a host of other troubles are promoted, if not, indeed, actually caused, by microbic toxins, but I differ from him very decidedly in the situation in which these toxins are in the majority of cases developed or produced —in other words, I believe the mouth is many times more frequently than the intestine the situation to be suspected. Again, Mr. Lane ascribed, in my opinion, much too active a *rôle* to abdominal adhesions in the production of intestinal stasis. I have on numerous occasions, both in print and in oral demonstrations, drawn attention to the exceeding frequency of these adhesions as found on the post-mortem table, a frequency many times over exceeding the occurrence of clinical symptoms which could even as a remote possibility suggest the performance of a colectomy. I certainly agree that these are a cause of alleged dyspepsia, but I cannot accept the view that they are either a cause or a consequence of a necessarily severe degree of constipation, such as—at least I take it so—must be the meaning of Mr. Lane's words, "intestinal stasis," though he has referred the Society to his published works for a definition of the condition. Dr. Jordan's slides interested me very much, but again I trust he will not think I am unnecessarily critical when I say that without a corresponding number of photographs taken from the normal subject the greater portion of what he has shown us is quite useless from the point of view of making deductions as to what is abnormal; the slides shown merely indicated shadows, but without the aid of stereoscopy it is quite impossible to accept the deductions as to the exact few inches of the bowel in which the kinks appear. A normal loop might easily appear an abnormal kink. Finally, as to the operation Mr. Lane suggests as necessary to obviate the trouble. In February of this year I saw one of Mr. Lane's old patients, and she was then as bad as ever, though he had operated on her some two or three years before. I would not use this case as an argument against any operative measures, but it does cause one to wonder whether

something short of a colectomy might not be tried. We must remember that a colon once removed cannot be replaced, and I would venture the suggestion that in cases of severe constipation in which all medicinal measures have failed it might be worth while to try what a simple appendicostomy would do. It is not such a severe operation, and I can vouch for the excellent results to be obtained, as I know of three or four patients of my own who are now, five or six years later, using the appendix for a daily wash out of the cæcum and colon.

Dr. E. VON OFENHEIM said that for the solution of the question of intestinal stasis it would be important to approach it from two different points of view—from the clinical as well as from the bacteriological one; but it would certainly be a great mistake to separate these two ways of investigation, as a satisfactory result could only be obtained if they went hand in hand, and if the clinical observations were compared with the results of the microscope. Clinical investigations had made great strides in late years, chiefly owing to the eminent work of Mr. Lane, but he was afraid that the bacteriological side, in spite of the enormous amount of work which had been devoted to the subject, had not kept pace with the macroscopical results, and that, taking it all round, their knowledge of the intestinal flora was a very limited one. Still more limited than their knowledge of the bacteria themselves was that of their action on the animal body and on each other. The question whether the presence of bacteria was necessary for animal life, as it was undoubtedly for vegetable life, was a very old one. It was Pasteur who first expressed the opinion that animal life was impossible without microbes. There were a good many followers in this theory, amongst whom he would only like to mention Schottelius, but also numerous opponents who had tried to prove by so-called aseptic breeding that animal life was possible without the help of bacteria. Amongst the prominent men who were of this opinion one had to mention Metchnikoff, Nuttall, Thierfelder, Cohendy and others. The experiments which were done in this direction were extremely difficult, and, so far, not conclusive, partly because they were carried out on domesticated animals, which meant animals living under conditions which were very different from their usual ones, and partly because the intestinal tract of these animals was anatomically very different from that of the human being. It was more important when it was found that certain animals had also in their natural surroundings an intestinal tract which was practically free from bacteria. Such a condition was first discovered in certain caterpillars of the species *lithocolletis* and *nepticula*. Amongst the mammals also Metchnikoff discovered certain species of which the digestive tract was practically sterile. One of these, a bat, named *Pteropus medius*, was most carefully investigated, but at the same time one ought to mention that this animal possessed practically no colon. Its digestion was much quicker than in any of the higher orders, and apparently the speed with which the food material passed through the intestinal tract was proportional to the size of the intestines. Only in the higher orders one found that the colon formed a large reservoir in which the unassimilated remains of the food accumulated and had time to undergo a process of decomposition under the influence of bacteria. In spite of this fact intestinal stasis was apparently unknown amongst the wild living animals; at least, he had never found any record that animals had been found dead owing to any sort of intestinal obstruction or any condition which would point to intestinal stasis. The interesting fact which Mr. Plimmer had reported concerning his investigations on animals in captivity could not be taken as an argument against this view, as one must not forget that these animals were living under conditions which were very different to their normal ones. Apart from the climate and the surroundings, the two most important factors were probably the lack of exercise —at least, compared with the violent exercise to which the majority of these animals were used to in freedom—and the quality of food, which, however carefully it might be selected, could not be the same as it was in their natural life. In domesticated animals, especially those which for many generations had been bred only from the point of view of their utility for the human race, and therefore were degenerates from the point of view of nature, intestinal disturbances and their immediate consequence (or should one say their cause?)—undue fermentation, were very common, and any veterinary surgeon would be able to confirm that the operation of paracentesis of the stomach in cattle for meteorism was almost as

common as appendicectomy in mankind. Amongst horses also, especially if they were deprived of sufficient exercise and were fed on damp grain, it occurred pretty frequently that the colon had to be tapped on account of the great development of gas. To the factors of insufficient exercise—*i. e.* complete transformation of ingested food into muscular energy and the inappropriate composition of the food itself—there came in the civilised human being another one, that is, the abnormal upright posture on his posterior extremities by which the pelvis had been tilted by 90 degrees, and the intestines by the simple action of gravity were forced down into it, and had to a great extent lost the liberty of their peristaltic movement. It was easily conceivable that these factors were producing excellent conditions for the development of bacteria—*i. e.* they formed a tube with very little or no movement, moist and warm, and containing nutrient media. One could, therefore, quite well compare the large intestine of man with an incubator which he carried with him in his abdomen. Investigating the species of organisms contained in this incubator, it was found that there was a large number of kinds. Mannaberg in 1898 counted twenty-seven, but since then the number which had been differentiated was at least trebled by recent researches. Only the roughest outlines of the physiology of the bacterial flora of the intestines could be touched upon in the narrow limit of time allowed in a discussion. On the whole, one could divide up this bacterial flora into two large groups —the proteolytic and the saccharolytic. The proteolytic one, as its name indicated, had the property of splitting up albuminous substances into various nitrogenous components, a good many of which were known to be poisonous. On the other hand, the saccharolytic had the power to ferment carbohydrates producing acids, alcohol and various gases. Amongst the first group he had to mention the *Bacillus lactis aërogenes*, *Bacillus perfringens*, *Bacillus sporogenes*, the *Bacillus putrificus*, *proteus*, the well-known *Bacterium coli*, and many others. Amongst the saccharolytic he only wished to mention the *Bacillus bifidus*, the *Bacillus acidi paralactici*, all the lactic acid bacteria and a great number of anaërobic ones. Between these two groups of bacteria existed a considerable antagonism, and no doubt we owed our lives to this constant fight between the groups, so that we could actually consider ourselves as being the *tertius gaudens* in this warfare. If the one or the other of these groups gained the upper hand it was man who suffered for it. Only a few of the metabolic products of the saccharolytic group were poisonous, and these only to a very slight degree, and it required an overwhelming quantity of them to produce any pathological symptoms. The products of the proteolytic group, the toxalbumins and ptomaines, were highly poisonous even in small quantities. Everyone knew the rapidity and disastrous course which so-called ptomaine poisoning took, that is, in cases where a large amount of these bacterial products is introduced into the system. There could not be any doubt that toxins were constantly produced in the intestines, in spite of the statements of Finkelstein, Falloise, Nobecourt and Rivet that the contents of the large intestine contained very few of these toxins in comparison with the contents of the small intestines. The fact which they had overlooked was that most of these toxins were either already absorbed in the small intestines or annihilated, or, more correctly, neutralised, by antitoxins and by the ferments of the saccharolytic group. Various arguments had been brought forward against the possibility of absorption of toxins through a healthy intestinal wall, the strongest of which were, in his opinion, the following: (1) That all cells of the body had the property of acquiring a local resistance against the action of poisons—the so-called local or cell immunity which one would expect would also be conferred on the endothelial cells of the mucous lining of the intestines under the influence of the toxins which were produced in them. (2) That the system reacted to the introduction of toxins of all sorts by the formation of antitoxins which, if the toxins were not in an overwhelming quantity, were able to overpower them. If one thought that absorption under these conditions was impossible, one must consider whether the condition of an uninjured normal intestinal wall ever existed in the civilised races. Surely in the majority of individuals it did not. First, because of the pressure and relative inertia of the large intestine which caused congestion and passive hyperæmia of this organ. Secondly, because there were constantly undigested particles of the food, like cellulose and mineral substances, passing through the digestive tubes which were liable to

cause slight abrasions of its walls. There could not be any doubt that toxins could be absorbed through the pathologically altered mucosa, and it was a well-known fact that many of these tox-albumins had a paralysing effect. One only needed to think of the clonic and tonic contractions in infants, commonly called "fits," which in a large proportion of cases were cured by a dose of castor oil or calomel, and which were simply a symptom of some toxic absorption from the bowels, to which the baby, with its deficient mucous membrane and undeveloped resistance, was more prone than the adult. The paralytic effects of the diphtheria and tetanus bacillus were also only too well known. Mr. Plimmer had told them that a rhinoceros which he cured with sodium sulphate and calomel fell ill with symptoms of paralysis and rigidity of the muscles—a condition which was frequently a premonitory symptom of colic in horses. Apart from their paralysing action, toxins had an irritating effect on serous membranes. Hay fever was an instance in which the mucous membranes of the nose and the conjunctivæ were irritated by pollen toxins, and the organic poisons of some jelly fishes were able to cause inflammation even through the epidermis in the same way as the stinging-nettle. Was it to be wondered at if the toxins of the intestinal organisms caused an irritation, or possibly even a local inflammation on that highly susceptible serous lining, the peritoneum, which responded to it by throwing out fibrin and forming adhesions which gradually stretched into bands? Mr. Lane called these adhesions "non-inflammatory." Could he prove that they were not inflammatory in their origin, although no signs of inflammation were to be found by the time they came under observation? That they appeared so frequently in the same positions could not be taken as an argument against this conception, as it was only to be expected that they would form chiefly in those parts which were more apt to be damaged and which formed a *locus minoris resistentiæ*—that is, where the strain and the attempt of the bowels to overcome any obstruction was greatest; as in the last coils of the ileum, the cæcum, the hepatic and splenic flexures, and the sigmoid. It could easily be understood that the paralysing effect of the toxins, together with the formed adhesions, were apt to retard or even partly to inhibit the peristaltic movement of the intestines and in this way to increase the amount of stasis. Thus the vicious circle was closed. Stasis due to unnatural conditions of life causing toxic absorption—the toxins causing increase of stasis. The wonderful mechanism of our systems tried to overcome the deleterious effect of the toxins, partly by excreting them, partly by the production of antibodies of various sorts. Transformation and excretion were done by the liver, the kidneys and the sweat-glands; and consequently those organs in cases of auto-intoxication were overworked and damaged. Hence the pathological changes found in them, such as biliary and urinary disturbances, hyperidrosis, etc. Antibodies again were produced in the ductless glands, the thyroid and suprarenal glands, and possibly also in the prostate. Metchnikoff had drawn attention to the *rôle* which the prostatic gland possibly played in the fight of the body against intestinal intoxication, and it might be that the absence of this organ and its secretion in the female body offered an explanation why auto-intoxication was so much more common and its effects far more disastrous in women, and especially unmarried women, than in men. If a great strain by a continuous and long-standing fight against the toxins was put on these various glands, it was within the law of Nature that a hypertrophy took place, and that the increased glandular tissues not only produced the *necessary* secretions to counterbalance the constant poisoning, but also a larger amount of those other secretions which normally and in a small amount were necessary to keep up the chemical equilibrium, but which in too large an amount over-balanced this equilibrium, and produced pathological symptoms such as occurred in "Graves' disease." The remarkable pigmentation of the skin in people suffering from intestinal stasis had, perhaps, something to do with a similar action of the suprarenal glands. It was also possible, although this was a mere hypothesis, that different kinds of toxins acted on different kinds of glands, and this might be an explanation why in one case one organ was chiefly affected, in other cases others. The field which lay open to research in this direction was enormous, and it would take a long time before all the darkness that still reigned over this subject would be cleared up. As it had been shown that the intestinal walls under certain conditions were permeable to toxins, the question arose whether they were

also permeable to the bacteria themselves. It was probable that what had been said of the toxins applied also to the bacteria—*i. e.* that the uninjured mucous lining of the intestinal tract formed a strong barrier to organisms, but that the slightest damage in its continuity would allow an invasion by them. The first conception of this idea they owed to Behring and Chauveau, who showed that the chief source of tuberculous infections in the infant was the intestinal tract, and this was easily understood if one bore in mind that the infantile intestinal mucosa was not a coat with a continuous and perfect covering. This only developed at a later time of life. Also, in the adult the continuous covering, as had been seen, might get damaged and open the door to a spreading infection, and as intestinal stasis was the cause of such damage, this could, perhaps, explain why so many various infections, amongst which must also be enumerated tuberculous infection, occurred in individuals suffering from chronic intestinal stasis. As well as tuberculous infections, bacilluria, cystitis, endometritis, pyorrhœa, and many other conditions were found associated with intestinal stasis. The question had been raised whether these infections of remote parts with intestinal organisms took place directly or through the blood-stream or lymphatics. It appeared to be of much less importance to find the lines on which they travelled than to know that the infection in these cases took its origin from the intestinal tract. Having shown that intestinal stasis, auto-intoxications, and bacterial infections formed a vicious circle, the question arose where and by what means they might be able to make a break in this circle. The first idea which presented itself would be to return to the natural conditions of life—a proposition which would at once be recognised as impossible to carry out in spite of the various attempts which had been made in that direction by the "simple life" enthusiasts and other cranks. The next attempt which could be made in the therapy of intestinal stasis was diet, eliminating those foods which were liable to form a good nutrient material to the inimical organisms in the intestines, and although this treatment might be efficient in some cases it certainly could not hold good in all, as the nature of the organisms which were at work differed so much, and the complaint could not be attributed to one particular species or even group. The aperients used for the purpose of increasing the peristaltic movement must be looked upon as a double-edged sword, as they were to a certain degree poisonous for the system and were acting as a strong stimulant to the sympathetic plexus, which after a time, unless the doses were constantly increased, got immune to this stimulation. With the antiseptic era the suggestion was raised to disinfect the digestive tract by antiseptics. Naphthol and tannic acid compounds were largely used for the purpose, but it was evident that it would be impossible to disinfect a tube with such rough and rugged walls as those of the intestines unless such considerable quantities of antiseptics were used as to damage the host more than the inhabitants. Lately paraffin had been suggested for the purpose—an idea which was by no means new. He had been informed that the negroes in South America used the ordinary paraffin for the treatment of intestinal troubles more than twenty-five years ago. As a matter of fact, in certain mild cases paraffin gave very satisfactory results—partly owing to its high bactericidal power; partly to its lubricating effect, by which it reduced considerably the strain of the intestines; partly, also, by covering the mucosa with a greasy coat, thus preventing the absorption of toxins. In serious and advanced cases it apparently had very limited or no influence, and when given for any length of time it lost its effect also in the mild cases, probably on account of the bacteria getting immune against it. This was what he had found in his limited experience of a few hundred cases. The next step which had been taken, and which, so far as he knew, was first introduced by Mr. Lane, was to destroy the haunts of the offending bacteria by a surgical operation by eliminating that useless incubator which had already been mentioned—the colon. This method had, so far, given the most satisfactory results in very advanced and almost hopeless cases in which all other measures had failed. Another attempt to make a break into the vicious circle, as previously mentioned, was made by Metchnikoff, Quincke, and others, which was based on the well-known antagonism that existed between the different groups of intestinal organisms. Certain bacteria were isolated, of which it was known that they were able to inhibit the growth of the dangerous species, partly by the amount of acid they produced, partly by their ferments. Two organisms were chiefly used for the purpose—the *Streptobacillus acidi lactici bulgaricus* and the *Bacillus bifidus*. In some instances, no doubt, good results were obtained, and although probably the principle of this form of treatment was a correct one and opened the best chances for the future, it had at the present moment two very serious drawbacks. The one was insufficient knowledge of the intestinal flora, so that it was exceedingly difficult, and sometimes even quite impossible, to find the antagonists of the detrimental organisms which were at work in every single case. The other and greater difficulty which would still have to be overcome was that, although it seemed such an easy matter to intro-

duce bacteria into the system, it was, in a great number of cases, and particularly in those which were highly toxic, very difficult, and sometimes impossible, to acclimatise the beneficial strains of bacteria. Extensive researches and experiments would be necessary before one would be able to overcome these difficulties, but one must hope that science and research would succeed in finding the ways and means to counteract the damage which civilisation had done to the health and welfare of humanity.

Mr. L. E. BARRINGTON WARD said that his experience with this condition had practically been limited to children. Stasis occurred as a disease in itself, and was by no means uncommon, but attention was much more frequently directed to it by the secondary affections that followed. The primary condition was often overshadowed by the secondary, and in the past had been neglected. When once constipation had been established, whether it was the large or the small intestine that was at fault, a chronic toxæmia was produced which caused definite changes. The tissues were devitalised, the resistance of the body was lowered, and the intestines became pervious to many organisms. He was inclined to believe that delay in the small intestine was more harmful to the individual and gave rise to a graver degree of toxæmia than delay in the large bowel. Hirschsprung's disease was almost a caricature of large intestinal stasis. In two cases which had recently been under Mr. Lane's care at the Hospital for Sick Children two or three weeks elapsed between the motions. These cases showed the general toxæmic signs well—poor circulation, low blood-pressure, pigmentation, etc.; but the effects were not so extreme as one would have expected. The explanation of this lay, he thought, in the fact that the bismuth radiograms showed no delay in the ileum, and at operation it was found that the distension and hypertrophy of the bowel were limited to the lower part of the colon, became much less when traced backwards to the cæcum, and were non-existent in the small intestine. Acting on the assumption that the constipation was the predisposing and aggravating factor in tuberculous infections in the child, Mr. Lane had short-circuited advanced cases of tuberculous joints which did not respond to prolonged treatment with the generally accepted measures. In the majority of instances the results had been excellent. Cases which had been treated by prolonged immobilisation under good conditions, which had been submitted to arthrectomy without success, which had persistent sinuses, and which were going fast downhill, had responded to ileo-colostomy, and were now, one or two years after operation, at home with the sinuses healed and the tuberculous disease quiescent. So bad was the condition in two of these cases that amputation to save life had been decided upon, but the improvement after short-circuiting the intestines rendered it unnecessary. They had had one very striking case of rheumatoid arthritis treated by this operation. This was a girl, æt. 10½ years, who had been crippled for two years and bedridden for three months with ankylosis of all the larger joints and many of the smaller. She had all the characteristic features of intestinal toxæmia. The usual treatment for rheumatoid arthritis had been given a full trial elsewhere, but she was becoming progressively worse. In October of last year ileo-colostomy was performed. All her symptoms improved at once. She gained ½ st. in the two months following operation, and when last seen, twelve months after operation, her weight had increased by 2 st. 4 lb. She had recovered full use of all her joints except the cervical, occipital, and wrist-joints, which had undergone osseous ankylosis. There were many other diseases which had intestinal stasis as their predisposing cause. In every case of recurrent appendicitis in Mr. Lane's ward stasis had been shown to be present by bismuth radiograms. Ulcerative colitis and *Bacillus coli* infections were other examples. From the breast-fed baby upwards constipation, shown by the infrequency of the motions, was exceedingly common. There was a great variation in the peritoneal relations of even the youngest children. In some cases the entire large intestine floated freely on a well-developed mesentery; in others the bowel was fixed back to the abdominal wall at various points or throughout its whole length. Such variations must have a profound influence on the functions of the tract, and explain why in some cases constipation occurred. When constipation of any degree was established a vicious circle was set up. Possibly the drag of the distended gut and the assumption of the erect posture caused the development of further bands from the materialisation of the lines of stress; possibly mild inflammatory changes occurred in the neighbourhood and led to the formation of the various bands and adhesions to which names had been given. Anyone who examined a series of post-mortem abdomens of children would be struck by the variety of the peritoneal attachments. As regarded the treatment, mild cases responded readily to simple dietetic and drug treatment. In the more severe cases ileo-colostomy gave good results. The operation was practically devoid of risk, and was well borne even in the smallest children. Subsequent diarrhœa was unknown. In a few cases regurgitation up the descending colon had been observed. It was most apt to occur in bedridden patients who had lost the habit of emptying the rectum. It was only necessary to ensure a daily evacuation of the rectum, and with the assumption of the erect posture the regurgitation was cured.

(*To be continued.*)

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SEPTIC INFECTION VERSUS CHRONIC INTESTINAL STASIS: THE LEGALITY OF ILEO-COLOSTOMY FOR ARTHRITIS.*

By PETER DANIEL, F.R.C.S.,

Surgeon with Charge of Out-patients, Charing Cross Hospital; Surgeon, Metropolitan Hospital; Surgeon, Gordon Hospital for Rectal Diseases.

IN the 'British Medical Journal' of May 4th, 1912, Mr. Arbuthnot Lane again urges the importance of what he once called constipation, but now calls chronic intestinal stasis, and its treatment by ileo-colostomy, with or without colectomy, but he has extended the treatment to cases of arthritis, in particular tuberculous and so-called rheumatoid arthritis. The subject is so important, that, for obvious reasons, it demands the most searching inquiry and investigation before this method of treatment can be adopted in a wholesale manner for its present purpose, namely, joint lesions. In effect Mr. Lane claims that all tuberculous and rheumatoid arthritics suffer from chronic intestinal stasis, but I would like to refer to a clinical fact which is of some weight, whether Mr. Lane is right or wrong, namely, that when we are informed by patients in our consulting rooms that their bowel acts only every second or third day we are greatly concerned; yet, put to bed, especially in hospitals, we find these same patients are *allowed* to continue these same habits without special efforts being directed to remedy it. The sight on the chart of a row of naughts with here and there a stroke is common; hence strict attention to the evacuation of the bowel is not a strong line usually in the treatment of our cases of, let us say arthritics, and if Mr. Lane's new application of ileo-colostomy does no more than forcibly direct attention to this great aid to treatment it will have done good. Yet, the able advocacy of this drastic treatment, whether right or wrong, by a surgeon of Mr. Lane's

* Delivered before the Medical Society of London.

eminence, invests the proceeding with such weight and authority as to frank it, in the opinion of many members of the profession not in intimate contact with intestinal surgery, as a presumably correct and accepted treatment for all cases of so-called chronic intestinal stasis, hence, also, probably of arthritis. Herein lies a grave danger, both to the profession, as being likely to be misled thereby, and, in particular, to the patients entrusted to their guidance.

It may be presumptuous on the part of one so unimportant as myself to question the accuracy of Mr. Lane, but I would ask leave to indicate, as somewhat extenuating my presumption, that since 1899, when I was surgical registrar to Charing Cross Hospital, I have consistently collected evidence anent what is called gastro-intestinal sepsis and its evils—local and systemic toxæmia. One of the first cases which clearly indicated to my mind its far-reaching influence on the body in general was sent in by Dr. Rowland Collum under Mr. Stanley Boyd's care (1899). He was submitted to laparotomy for urgent symptoms suggesting perforated duodenal ulcer, but was proved to be free from any peritoneal lesion. I soon after recognised it as a case of exacerbation of a chronic gastro-entero-colitis of oral septic origin.

In May, 1903, I read a paper before the Harveian Society, entitled "Gastro-enteritis of Septic Origin, without Diarrhœa, simulating and treated surgically as Peritonitis, with Notes of Four Cases." A brief abstract was published in the medical journals: the original paper I have before me now. I had records of other cases collected during the preceding three and a half years, but in these some of the various *complications* of intestinal sepsis had arisen, and I did not wish to confuse the issue presented by my paper.* In it the close causal connection of oral sepsis with the gastro-intestinal lesion was emphasised and the profound "toxæmia" recognised. I called pointed attention to the possibility of universal toxic and septicæmic sequelæ arising from the gastro-intestinal infection, and mentioned particularly nephritis, synovitis, endocarditis, parotitis, nervous disorders, and suggested a whole gamut of infective processes might arise in this way — *locally*, ulceration, peritonitis, intussusception, etc., thus, early recognising the ill-effects of what is called gastro-intestinal sepsis, what Mr. Lane calls auto-intoxication, which he attributes to stasis, but which certainly do arise from infection without the existence of stasis.

I showed that the lesions in the four cases quoted above were really *exacerbations* of chronic septic catarrh; that the absence of diarrhœa and presence of "almost" typical intestinal obstruction was, I believed then, peculiar to the *acute* lesion, as I believed the current teaching that *chronic* intestinal catarrh must be associated with looseness of the bowel, which I now know to be inaccurate.

It was not till a year later, 1904, that I was appointed to the staff of the Gordon Hospital for Rectal Diseases, and in time at the Gordon Hospital I learned from practical experience that chronic intestinal sepsis (*i. e.* "catarrh") was a very prevalent cause of constipation (I use the word in the sense now popularly accepted), and that from time to time such patients suffered from attacks of looseness of the bowel (hence the name "intestinal catarrh"), but that *persistent* diarrhæa only occurred when definite colitis, especially sigmoiditis, supervened. In short, I learned that sepsis first led to stasis.

From 1904 onwards I have kept vigilant observation on my Gordon Hospital patients (all intestinal cases), and am increasingly firmly convinced in my belief that the most potent factor in inducing *all* intestinal lesions commencing in the mucosa, and including constipation, is oral or nasal accessory sinus sepsis.

My conclusions were briefly indicated in an address delivered at Tottenham, and published in the 'British Medical Journal,' January 15th, 1910, also in some remarks delivered at the Odontological Society. I attributed to gastro-intestinal septic infection, in any part, the power to so damage and break down the resistance of the tissues of the body as to render them vulnerable to attack and invasion by any bacteria which might be conveyed to them in sufficient number or on repeated

* Since, I have been called to operate upon twenty-five such cases, but having learned how to suspect and recognise the cases as belonging to the class of infected mucosæ I have avoided the mistake of submitting them to an unnecessary operation. The great difficulty to decide is whether *in addition* there is a complication present, *i. e.* has the mucosal infection now progressed into an appendicitis or other entity demanding for its effective cure an operation?

occasions, but against which in all probability the tissues would have been invulnerable had no previous damaging "spade work" been performed by the gastro-intestinal infection and toxæmia.

In May, 1910, I handed in the MS. of my book on 'Arthritis: a Study of the Inflammatory Diseases of Joints' (Bale). In this work I indicate in no ambiguous terms the immense importance of septic infection of the gastro-intestinal tract as a predisposing cause of tuberculosis of joints, and conversely the importance of the removal of this sepsis in the successful *treatment* of the tuberculosis of joints. From pp. 25 to 29 and at p. 317 and elsewhere the case is elaborated, and at p. 389, in dealing with the treatment of tuberculosis of the hip, is stated: "At the risk of burdensome repetition, I must again state that the best results in tuberculosis depend upon the state of the gastro-intestinal tract, *the absence of sepsis therein* and in other parts of the body."

Similarly, to gastro-intestinal sepsis directly I attribute most osteo-arthritis, while the varieties known as rheumatoid arthritis (which, with tuberculosis of joints, Mr. Lane now deals with by ileo-colostomy) I consider are due to a *combination of* gastro-intestinal *and* genito-urinary sepsis. I have a female under my care now who bears out this view, and whose recovery is sufficiently marked to evoke such enthusiasm as Mr. Lane expresses for the recoveries made under the method of treatment by ileo-colostomy. She has intestinal stasis, but she *had* considerable oral sepsis and also genito-urinary sepsis, but I am pleased that the treatment was not so drastic as short-circuiting.

Before Mr. Lane wrote in the terms he now does on intestinal stasis, I had perceived that gastro-intestinal sepsis was a potent cause of joint inflammation—I called it "synovitis" (in the book on arthritis I discuss the inadequacy of the term "synovitis")—and was therefore a predisposing cause of tuberculosis of joints. I am not rushing into print therefore on a subject I have not studied, since I may truly say gastro-intestinal sepsis has been my constant study since 1899, and genito-urinary sepsis since 1902 (see "The Cause of Enlarged Prostate," 'British Medical Journal,' October 29th, 1904).

I am therefore at one with Mr. Lane in accepting the widely spread evils which may arise from the disordered gastro intestinal tract, but whereas in the main *he* considers the primary cause to be mechanically evolved bands and kinks which lead to stasis and the stasis in turn to auto-intoxication, *I* believe the evil is primarily an infection, *and recurring re-infection*, of the gastro-intestinal tract, and is in direct proportion to the number and virulence, also to the varieties, of the infecting germs, and that all the ill-effects Mr. Lane mentions may arise without any stasis in any part of the tract.

His point of view may be inferred from the term he uses, "the cæcum is the cesspool of the body," when in reality it acts as the second stomach where the food undergoes the most thorough mixing and profound digestion, and it is only when the food and intestine is infected with pus-forming and other abnormal pathological bacteria that it may fairly be called a cesspool, under which circumstances, even in the absence of abnormal stasis, it *would* act as a cesspool.

As Mr. Lane propounds a mechanical hypothesis of the formation of the bands and membranes which produce the stasis, I would like to point out that I am quite sympathetic with the desire to attribute to a mechanical factor, any explanation justly due to the influence of pressure, gravity and other simple forces, having myself expounded the influence of the mechanical, incorporated in a paper read at the Polyclinic in 1908 on "The Treatment of Tuberculosis by Iodoform Glycerine and Bier's Methods," under a subheading, "The Influence of the Physical or Mechanical in the Production of Disease and its Spread, and conversely in its Cure."* Hence I am not unacquainted with either of the two important elements in Mr. Lane's paper, namely: "Intestinal Toxæmia and Mechanical Factors producing Disease."

The etiology of Mr. Lane may best be described in a simple manner as "putting the cart before the horse"; the process is one of infection and re-infection of the stomach and bowel. The cæcum is the earliest and most severely affected part of the *intestine*, since *in the cæcum the food is meant to be delayed*, and there by a process of local peristalis and anti-peristalsis to be thoroughly churned up and mixed with the digestive juices, and only when the chemical consistence and character of the food is satisfactory is an onward peristalsis conveying the

* Peter Daniel, 'Medical Press,' November 11th, 1908, p. 518.

food into the upper part of the ascending colon and transverse colon permitted.

As a result of this normal delay (but not stagnation, remember) of the intestinal contents in the cæcum, the mucosa of the cæcum is subjected to continuous and prolonged insults from any bacteria present in the food. If the bacteria are few, and especially if they belong to what have been described as the normal bacteria of the intestine, few ill-effects accrue, and *little or no absorption of toxins* occurs, as will be shown (Murphy and Vincent).

But if the bacteria are numerous, and if constantly reinforced by virulent germs from suppurative processes present in the upper digestive tract and naso-pharynx, then the *cæcal mucosa is damaged*, lymphangitis results, the glands in the ileo-cæcal angle are infected, peritonitis occurs over the cæcum, the appendix and lymph-glands, and thickenings or adhesions form. *These thickenings are Lane's bands*, and, like all pathological fibrous tissue, contract as they get older, and I ask Mr. Lane to produce any pathologist of repute who supports his view and to refute this origin of the bands. The peritonitis localised over the cæcum which one constantly sees is called Jackson's membrane. Thus we find mucosal infection is primary, pathological bands secondary, and stasis may, or may not be, a sequence. The universal adhesions seen from time to time covering the whole of the intestines, and wrapping them in dense membranes, are the result of former tuberculosis, perhaps aided and added to in the way I have just described. Gastritis and duodenitis accompany the foregoing changes *from a common cause, not as sequelæ*, and by no stretch of the imagination can I conceive that Mr. Lane's reasoning is sound on the pathology of *these* so-called sequelæ of intestinal stasis.

Give these cases correct treatment, *i. e.* cleanse their mouths, naso-pharynx, antra, etc., so removing infection, give them oils, suitable laxatives, and diet, and the number of cases requiring colectomy will be at once reduced to those unfortunates in whom the damage done to the mucosa has progressed to ulceration.

We know that when the exciting cause is removed adhesions may disappear absolutely, and it is only rational to assume such occurs under the conditions I have briefly sketched out:

I am at variance with him as to the *origin* of the kinks and bands which he blames for the stasis. In common with most pathologists I attribute the formation of the bands, etc., to *local* peritonitis, the result of bacterial activity in the underlying hollow viscus or in the lympathic tract leading from and draining its wall, which necessarily implies that bacterial infection of the mucosa is primary; since absorption of toxins does not take place through the healthy mucosa,* the bands and kinks are secondary to this pathological absorption; stasis may, or may not, result from the infection, therefore may, or may not, *precede* the formation of the bands, etc.

Mr. Lane says the bands are non-inflammatory, are *primary* in point of time, and result in stasis, the stasis in auto-intoxication. If the bands form from abnormal "dropping of the viscera," why do the viscera drop? The bands are secondary, but in dropped intestines infection of the mucosa is likely to occur.

I am also totally at variance with Mr. Lane in the line of treatment he adopts in general, especially in its bearing on arthritis. (I do not deny the absolute need for short-circuiting in certain cases one is constantly confronted with, *i. e.* of *pathological* membranes and bands causing obstruction.)

I believe that if the infection is cured, *i. e.* the origin of the infection in the mouth and elsewhere is cured, the stasis in the vast majority of cases will tend to cure spontaneously, and if appropriately aided the patients entirely recover, *i. e.* they are damaged, but the stomach and intestine functionate sufficiently well, often as if the patient were normal; should bands or other grave pathological sequelæ have formed, the recovery will be partial and proportionate to the lesions. Apparently bands, obstruction and stasis occur in almost all Mr. Lane's cases of arthritis.

My meaning of the importance to be attached to the gastro-intestinal tract in the *treatment* of tuberculosis of joints is also that, if the tract is infected it must be made healthy (in conjunction with all other infected parts of the body), chiefly by cutting off infection, etc, but if already healthy or made healthy, then, instead of short-circuiting the large bowel and thereby depriving the patient of a highly important digestive organ, I would like to add a foot of cæcum and ascending colon to the existing intestine so that the additional nutrition so

* Murphy and Vincent (see later reference).

absorbed might be acquired to overcome the lesion in the joint. I do not for one moment believe any portion of our anatomy to be unnecessary or injurious to us.

Mr. Lane now denies the importance of oral sepsis as an additional factor in *increasing* the evils of stasis (see his former works). Does he not endeavour to remove all such sources of intestinal and systemic infection *before* he short circuits? Does he wait to see the improvement which invariably follows? Or does he "clean up" generally and short-circuit simultaneously, but gives *all* the credit for any improvement to his pet operative measure? Or do most of his cases have intestinal stasis without previous oral or other sepsis?

If gingivitis is the *result* of intestinal stasis, as Mr. Lane now states, does *it* clear up when colectomy is performed? By analogy it should do so, since cysts of the breast and thyroid "recede" and get quite well when the bowel is dealt with.

I would like to know whether interstitial mastitis in women who have lactated also recedes and disappears in this way? It is exceedingly hard to credit.

Having shown myself *en rapport* with Mr. Lane up to a certain point, let me discuss some aspects of his papers which may roughly be divided into four parts: (1) A summary of the evils of stasis; (2) the mechanical factors which result in stasis; (3) the symptoms of stasis—toxic, X-ray; (4) the treatment of stasis.

What is chronic intestinal stasis? and what is the substance, or substances, which results, and is the cause of auto-intoxication?

Definition of chronic intestinal statis.—Mr. Lane says: "By chronic intestinal stasis I mean such an abnormal delay in the transmission of the intestinal contents through some portion or portions of the gastro-intestinal tract as results in the absorption into the system of a greater quantity of toxic material than the organism is able to deal with effectively by means of the organs whose business it is to eliminate it."

This definition is in no way qualified; it matters not from the point of view of treatment whether the intestinal tract is acting as the sewer to abscesses in the upper gastro-intestinal tract or is free from this serious contamination, nor whether the patient has a long or a short mesentery. It is immaterial whether the primary lesion was some gastropathy developed in childhood, as is very common, especially consequent upon unsuitable diet* or some debilitating diseases or general infections, but in almost all cases of which gastritis is present.† For all, the treatment is: ileo-colostomy.

Nor is it material that constipation, produced by negligence and other simple causes, has existed for years with little or no obvious ill-effect; as long as a meal containing sufficient bismuth, as Mr. Lane somewhat naïvely puts it, shows retardation in its progress through the intestine, and the patient shows any evidence of general toxæmia (which Lane does not set about to prove is not due to other causes than gastro-intestinal), then the patient suffers from chronic intestinal stasis, and the only suitable treatment is ileo-colostomy.

Mr. Lane casually refers to the multiplication in the stagnant area of the pre-existing bacteria, "and that their level in the intestine is raised in a corresponding manner." What he means by this exactly is not clear, but in constipation the bacteria pre-existing in the fæces diminish in number. In diarrhœa they first increase, later they may diminish.

It is obvious from the whole tenor of his paper that what he lays stress upon is the delay in the passage of the "material," *i. e.* the food, and that the primary cause of this delay is purely mechanically developed bands, *the result of the erect posture assumed by man*, that the bands are primary in point of time, *i. e.* band first, stasis second. The usual pathological explanation of peri-colic bands and adhesions, namely that they are the result of intra-intestinal lesions and lymph-gland infection, *i. e.* intestinal lesion first, band second, stasis third, is incorrect, according to Mr. Lane.

Further, he thinks that the *mechanical* retardation of the food is the main cause of the various secondary lesions mentioned by him, as a result of simple *obstruction*; this is aided in some small measure by the toxic properties evolved by the *retardation* of the food. He ignores the important bio-chemical changes which may in themselves *cause* retardation in the onward passage of food.

All that Mr. Lane claims for mechanical stasis is much more rationally explained by the theory of intestinal sepsis, *i. e.* when the bowel really acts as the sewer for an infective or septic process taking place in the commencement of the gastro-intestinal tract. Because the bacteria are hurried with the food through the *small* intestine, it does not follow that they are inactive there, but absorption of their end-products occurs along the small intestine as certainly as it occurs in the cæcum and large bowel. But in no part of the mucous-lined tracts are toxins absorbed until definite damage, *i. e.* by inflammation, is done to the epithelial lining.

The mechanical evolution of bands.—It was my impression that the primitive man who progressed on all fours, or even arboreally, was an exploded myth; that being so, I fail to see that the evolutionary theory of Lane has any basis of fact; but, granted it were so, are we really to believe that the poor infant of two years of age, whose ileum was short-circuited, had already *evolved* a new band

* Cautley, 'Royal Society of Medicine.'
† Hayem, 'Journal des prat,' November 18th, 1911.

or membrane at his mature age? What may he not develop about his now abnormally situated ileum by the time he reaches man's estate?

Mr. Lane writes of a *simple* static stasis; are they not all simple according to his explanation, and are they not all static? What are the other varieties?

Other modern authorities explain visceroptosis, which must, logically, be the exciting cause of Lane's bands, differently, and without greater support than that yet given it, we cannot really accept Mr. Lane's explanation of the purely mechanically acquired bands, since he absolutely refuses to admit into the process the possibility of bacterial activity, even secondarily.

If the bands are evolutionary, *i. e.* physiological, why are they injurious? Evolution is always *helpful*, not injurious. If evolutionary, why are they not permanent in the human subject? Mr. Lane says they develop in children of two years of age. It seems strange that if so readily developed they have not become normal instead of occasional. *In utero*, the various translations of the large bowel resulting in the adult mesentery and ligaments are not influenced by gravity; on the contrary.

What does Mr. Lane mean by toxic material? Whence is it derived? In the case of food retarded in its passage through the intestine it is possible for toxins to be derived from (1) destruction of epithelium, (2) perverted digestion products—*true* auto-intoxication; (3) decomposition and fermentation of food; (4) the end-products of the life and activity of bacteria.

To which of these toxins does Mr. Lane attribute the blame for the secondary toxæmia? If there is much epithelial destruction in these cases of mechanical stasis it should be manifest in the excreta, and bearing in mind the work of Bolton and others, one ought to try to ascertain if there *is* such destruction. Has Mr. Lane tried to find out by examination of the fæces? It is stated that by skilful examination of fæces localisation of the actual *site* of intestinal lesions is possible, and since such expert pathological talent is at Mr. Lane's command it would be possible for him to obtain evidence in support of the views he propounds, *i. e.* if the evils of the stasis arise chiefly in the small intestine then the fæces should convey this information.

Does Mr. Lane look with the sigmoidoscope to see if there is evidence of disease in the sigmoid or rectum before he short-circuits? He does not state that these examinations are made, yet he does not hesitate on the evidence of an X-ray examination to short-circuit the bowel in a child of two years of age.

If *fermentation* is productive of toxæmia, then the use of the lactic-acid-forming bacilli so prevalent a short time ago and from which such good results were said to be derived ought to have added to the toxæmia of the patients.

With one exception all investigations on ptomaine poisoning show that it is highly probable the chemical bodies resulting in so-called ptomaine poisoning are the end-products of certain pathogenic bacteria and *are not derived from decomposition;* which supports the conclusions of recent researches, which all point to the end-products of bacterial life as being responsible for intestinal toxæmia.

Clinical evidence is also against the view which formerly attributed to perverted digestion products the origin of the great "dyscrasiæ," and increasingly attributes them to the toxic action of bacterial processes, especially when involving the gastro-intestinal tract. Certain experiments I quote (see later).

One assertion of Mr. Lane's I have already mentioned, namely, that "as a result of stasis the number of deleterious organisms in this (the stagnating) segment of the drainage system is increased, and their level in the intestine is raised in a corresponding manner." All evidence is to the contrary in constipation, which affects the big bowel, and it is reasonable to conclude the same occurs in stasis in the small intestine; at any rate Mr. Lane adduces no support to his statement.

In "An Experimental Study of the Cause of Death in Acute Intestinal Obstruction," Murphy and Vincent* state there are three princip theories:

(1) The theory of direct transmission of bacteria through the wall of the obstructed intestine, causing a *fatal peritonitis.*

(2) Nervous reflex, which attributes the death to collapse, etc., from irritation of the nerve-filaments of the intestinal wall.

(3) The theory of auto-intoxication, *i. e.* the absorption of some material from the obstructed bowel; this substance is due either—

(*a*) To the activity of the micro-organisms which multiply in the obstructed bowel, or—

(*b*) The essential factor is some disturbance in the *secretion* of the intestine itself.

(1) The first theory is put out of court by the non-development of peritonitis and the low toxicity of the free fluid found in the peritoneum even in the terminal stages.

(2) There is no positive evidence forthcoming to support the nervous reflex theory.

(3) They state that the toxic intestinal contents are not absorbed to any degree through *normal* mucous membrane; hence, when the obstruction is removed and the toxic contents pass on into the unobstructed portions no absorption of the chemicals or decomposition products takes place.

They have summarised their work thus:

* Murphy and Vincent, 'Boston Med. and Surg. Journ.,' November 2nd, 1911.

(1) Interference with the circulation of obstructed bowel is the vital factor in the production of the typical symptoms of acute ileus. The obstruction to the venous return is the most important element in this circulatory disturbance. (This causes one to inquire how much the mere stasis of intestinal contents, which it is not stated is accompanied by vascular disturbance, produces in the way of toxins?)

(2) The responsible toxin is destroyed by boiling. It is not soluble in water, and will not pass through a Berkefeld filter.

(3) The formation of this substance is not dependent, probably, upon any vital *secretion* of the mucous membrane of the intestine.

(4) The rapidity of absorption varies, dependent upon the patency of the lymphatic channels in the mesentery and the permeability of the intestinal wall.

Conclusions.—"We conclude, therefore, that this toxic substance is purely bacterial in origin, *and that the living bacteria with their end-toxins*, not the putrefactive products nor the chemical poisons, are directly responsible for the profound symptoms and death in acute intestinal obstruction."

Their conclusion, in fact, is that the bowel acts simply as a test-tube, but one which not only houses the bacteria, but permits the removal of their effete products, *their end-toxins*, to which fact is due the symptoms of toxæmia. The fact that they are in the food tract is an incident; they might as well be in a muscle or antrum in so far as systemic poisoning — so-called auto-intoxication — is concerned.

Other experiments have shown that even a linear band of necrosis in the intestinal wall may result in death without the intervention of intestinal obstruction, *i. e.* complete stasis. I have reported * two such cases of death from linear necrosis due to pressure exerted by the tight edge of the aponeurosis of the external oblique when the incision in it for colotomy was too small, in neither of which cases was stasis of the bowel contents permitted as they were washed out daily. Hence in them the toxæmia could not be attributed to either putrefaction or fermentation of the food, nor can one attribute it to perverted function of the secretions. Post-mortem there was evidence of bacterial activity, in the one case strictly confined to a linear circumferential necrosis one and a half inches from the severed end of the gut.

Whether we are justified in applying these experiments and clinical observations to *chronic* intestinal stasis I leave to the reader; they seem to me to bear directly on the subject, since they all credit bacterial activity with the greatest share in production of the toxæmic symptoms, and imply that mere stasis is implicated only in so far as it increases or conduces to the activity of the bacteria.

In so far as stasis in the colon, *i. e.* constipation, is concerned, every clinician knows of instances of marked constipation of many years' duration who do not appear to show any ill-effects of the stasis, yet no one will deny the intense gravity and fatality of these same cases once bacterial invasion of the mucosa has occurred. Ulcerative colitis has a direct mortality of about 70 to 80 per cent.

Hence all evidence suggests that the grave constitutional symptoms which Mr. Lane attributes to stasis, a mechanical condition, are due in truth to absorption of bacterial end-products, and we *know* such absorption does take place *without stasis*, and then produces just such symptoms as Mr. Lane credits to mechanical stasis.

Mr. Lane lauds the receptivity of American surgeons: I am entirely with him, but does that help at all? Gasserianectomy has been performed more extensively in America than in any other country probably, certainly more frequently than in England, yet we are told by Mr. Lane himself that this operation is quite unnecessary, as the symptoms are due to intestinal stasis and the proper treatment is colectomy; hence mere receptivity is no argument. Nor is it necessary to go to America, where many surgeons and others seem to be just a little too "receptive," for corroboration of this statement; it has become overwhelming in this country, although in no country do the inhabitants suffer more from intestinal toxæmia than in America, where oral and nasal sepsis is artifically cultivated by the dentists to a degree hard to estimate.

The majority of American surgeons and pathologists, however, fail to accept the purely mechanical origin of Lane's bands, etc. Pilcher,* who is thoroughly alive to the advances in surgery, and quotes Jackson and Lane amongst others on this point, as recently as 1912 says: "The most probable explanation of the origin of these bands and membranes is that they are the result of long-continued or oft-repeated mild infections of the peritoneal covering of the cæcum and appendix" —a statement I thoroughly endorse.

My belief is that typhlitis† is the earliest lesion to arise in the case of intestinal sepsis, because the normal retardation in the passage of food which occurs in the cæcum permits of a great concentration of the bacteria swallowed, and it is therefore especially subjected to toxic insults in which the peritoneum covering it participates, and sooner or later the appendix with its mesentery and their peritoneal covering is involved, and from their juxtaposition the two peritoneal layers readily cohere, leading to kinks, etc.

In fact it seems to me that Lane's short-circuiting

* Peter Daniel, "Colotomy and Some Misconceptions of its Results," 'Lancet,' November 18th, 1911.

* Pilcher, 'Ann. of Surgery,' January 12th, 1912.
† Peter Daniel, 'Proctologist,' March, 1912.

operation is a practical proof of my contentions; he drains a septic tract and permits of a quicker exit to bacteria, thus shortening the period in which they may produce end-toxins; but instead of cutting off the source of supply and rationally treating the stasis, Mr. Lane out-rivals Paddy, who burns his house to obtain roast pig.

Objection might be made by Mr. Lane that constipation is not what he means by chronic intestinal stasis, yet this is what he first called it. Let it be granted that he refers to small intestine stasis; there is no doubt that colon stasis is a more toxic producing process than stasis in the small intestine, as the bacteria are relatively fewer in the small than in the large intestine (a film of the Pathe Frere's shows this clearly), while in the latter also the food has become fæcal.

Hence colon stasis as stasis, *i. e.* retention of food, should be more injurious than ileal stasis, yet we know clinically that such chronic stasis may exist for years with little or no apparent ill-effect; * but if Mr. Lane will grant that the whole cause and effect, bands, membranes, and the associated lesions, is a matter of prolonged inflammation, by contamination of the whole gastro-intestinal tract by bacteria, and bacteria of a very virulent nature, because they are constantly *reinforced* by new colonies of staphylo- and streptococci habituated to the tissues of the host, then we can understand the whole scheme of chronic intestinal stasis, and why these parts of the tract where so many lesions are most prevalent are also the site of bands and membranes, and give all credit due to every accessory factor, gravity, *i. e.* the erect posture, included.

There is one remark by Mr. Lane with regard to the method of examination which merits attention: it is, "that a sufficient quantity of bismuth carbonate must be given." To the uninitiated this seems to suggest that if you do not give enough bismuth the opacity of the meal administered will not be dense enough to produce a shadow, but this is not the meaning Dr. Jordan gives, in his papers on Lane's kinks.† The ordinary plan adopted is to give a pint of well-prepared broken-up bread in milk, free from lumps and grit and made palatable according to taste; in this a suspension of 2 oz. of purest bismuth carbonate is stirred. (Mucilage is unpleasant to the palate and should be avoided.) Jordan says: "With 2 oz. of bismuth in the meal the whole of the bismuth is in the cæcum and ascending colon at the end of six hours in most normal persons; *if we double the dose*, and still more if we increase the dose to 5 or 6 oz., we find some bismuth in the lower end of the ileum after five, six, or even eight hours, even in normal persons, and we have a far better opportunity of detecting slight abnormalities in this region, while we attain much truer pictures in well-marked cases than we could possibly obtain with the customary 2-oz. doses."

According to Jordan's logic, if the dose of bismuth is increased sufficiently bismuth will be found in the lower ileum of normal persons twenty-four hours or more later. In view of the very far-reaching deductions drawn from stasis of bismuth by Mr. Lane, it is necessary to remember the foregoing findings by simply increasing the dose of bismuth.

Landsberg* says "the ingesta remain an extraordinary long time in the cæcum and ascending colon, as long as twenty-four hours in normal persons," and Jordan says "that bismuth retained for long in the ileum becomes dried up." The bearing of this on the chemistry of digestion must be considered. We know that food is subjected to a very thorough mixing by peristalsis and anti-peristalsis in the cæcum and ascending colon, and that until its consistence and chemical condition are satisfactory its onward progress is delayed. Landsberg says "the consistence of the contents obviously plays a chief part in deciding when anti-peristalsis is replaced by the forwardly directed peristalsis."

Most recent research tends to increase the relative importance of the disorganisation of the chemical factors and of secretion and absorption rather than motor weakness or mechanical difficulties of a slight nature in causing constipation, *i. e.* stasis; but Jordan says "the form of the test-meal for ileal examination is not important," and that the changes which lead to Lane's kinks "consist of a general dropping of the cæcum," yet the two pictures selected to demonstrate this show nothing of the kind. Fig. 1, Pl. II (reprint), shows a cæcum normally situated, while fig. 2 has a high-lying cæcum. Is it possible, therefore, Jordan's results are in the main artificial? The collection of 4 or 6 oz. of bismuth in a cæcum and ascending colon is likely to embarrass both the chemistry and motor functions of the best regulated intestine, seeing that the weight of a normal stool (the collection of twenty-four hours' food, not of one meal) is only 6 to 8 oz.

Most authorities state that kinks of the intestine are associated with excess of mucus in the stools, also carcinoma of the cæcum results in mucus (and blood). Is there any evidence of mucus in these stasis cases?

In the case of a somewhat delicate woman the whole excreta of twenty-four hours often is less than 4 oz. in weight, and we are asked to believe that the sudden ingestion of 4–6 oz. of bismuth—dry, sedative, impervious to the juices, and very heavy—will be passed along the bowel at the

* Peter Daniel, "Idiopathic Dilation of the Rectum, with Notes of Two Cases," 'Internat. Clinics,' vol. ii, 1909.

† Jordan, 'Brit. Med. Journ.,' May 20th, 1911; 'Practitioner,' April, 1911.

* Landsberg, 'Wien. med. Klin.,' No. 27, 1911.

normal rate, and have no influence on the mucosa. It is not reasonable.

Dr. Jordan also stated that when oleum petrolei was administered to the patients with intestinal stasis, "the evidence of stasis was not obtainable," *i. e.* the bismuth emulsion passed into the cæcum and did not collect in the ileum.

This seems to me to give away the whole case in so far as bismuth X-ray diagnosis is concerned.

Whether the oil mechanically helps the bismuth through the ileo-cæcal valve or whether it prevents that *inspissation* of the bismuth which Jordan says takes place in the bismuth meal when it is delayed in its transit is not yet clear, but in my opinion the oil does various things which are beneficial; suffice it that the oil effaces "the evidence of stasis."

One would conclude that if such evidence disappeared the need for operation ceased. But no; we are informed—"At the subsequent operation the usual appearances were seen," etc.

With regard to duodenal obstruction, Jordan makes this observation: "This case gave me a hint: it was clear I should only succeed in demonstrating abnormalities of the duodenum while there was obstruction to the passage of food through it"; the impression one gets on reading the paper is that he can only get the pictures he wants when he can get a *large bulk* of concentrated bismuth meal *quickly* into the duodenum.

He says: "The test-meal must not contain lumps lest they set up pyloric spasm, which prevents the *rapid* filling of the duodenum—*a necessary condition* to the demonstration of the phenomena of duodenal distension from delay due to duodeno-jejunal kink." Surely it means that if the bismuth meal gains admission gradually, as we are led to believe is normal, no distension occurs, otherwise obviously the opaque bismuth would show.

If the actual test-meal which forced this conclusion upon Mr. Jordan had gained admission to the duodenum gradually would it have been at all delayed?

He says: "To effect the sudden filling of the duodenum I find it necessary to give a full-sized meal containing 4 oz. of bismuth carbonate in the form of a thick emulsion; . . . it is usually necessary for the patient to lie on the right side for two or three minutes to enable a large proportion of the meal to enter the pyloric part of the stomach and to pass through into the duodenum." If this statement is true of what actually occurs in a patient then the stomach in question must be abnormal, and the case suggests that the duodenum is overcharged by too free entry of food, not by obstruction at its outlet; a distinction of vast importance.

He continues: "Successful discovery of duodenal obstruction depends almost necessarily upon an unobstructed pylorus; when the pylorus is obstructed or the stomach is not contracting actively the duodenum never becomes properly filled and the characteristic appearances are not obtained." Of course it all depends upon whether what Jordan calls "filled" is what occurs naturally. Yet most of the instances he portrays have dropped stomachs, which clinically we know means delay in emptying themselves, and results in the same thing as defective contractility, which is so fatal to success, yet he states specifically that there is no delay in these cases in emptying themselves but contradicts himself in his papers.

Also after stating that the *bismuth* meal must be present in the duodenum in bulk before the characteristic appearancesare obtained, he says: "At the end of fifty hours powerful contractions of the duodenum were seen to be taking place, altnough it contained nothing more opaque than Benger's food." Then what does this mean?

Continuing anent the difficulties of obtaining a "demonstration" of duodenal obstruction: "Hence I was most successful with ambulatory patients, and with these only when they were in pain, showing that their pain was due to obstruction," *yet most of these cases are always relieved by taking food.*

In Jordan's illustrations one of the striking features is this: In all the duodenal cases the shadows are confined to the right side of the spine: if this is so it follows that the real third stage of the duodenum which crosses the spine to the left side to become the fourth stage, which then joins the jejunum to the left of the midline, these two parts, measuring some five inches, contain *no* bismuth, else they would give a shadow; certainly they are not *distended*, if they are not actually empty. How comes it that an obstruction at the outlet of the duodenum (duodeno-jejunal flexure) does not cause stasis in the near third and fourth stages, but does produce stasis in the distant first and second stages?

Is it not probable that the apparent distension in the first and second stages is but an exaggeration of the normal stasis destined for the more perfect admixture of the food with the bile and pancreatic juice?

If any mechanical factor is necessary to account for stasis in the first two stages is it not more accurately attributed to the pressure or traction exerted by the transverse colon, which here crosses and is intimately attached to the duodenum, particularly when it is loaded? Also what part may a dropped kidney play in the pathology.

If the obstruction is purely mechanical why is it that Jordan finds it intermittent? He means that he can only demonstrate it intermittently. Lying down will not explain this intermittency, as he says later—"the distension of the first stage of the duodenum may persist and be demonstrable during the performance of an operation," despite the posture. Also—"the distension may not be suffi-

cient to produce any change in the duodenum; it may, however, lead to engorgement, or in more severe cases to ulceration of the mucous membrane." If distension produces ulceration, surely it must be by interfering with the vascular supply, especially of that special portion of the first stage which is supplied by the "supra-duodenal artery" (Wilkie*)—a small end-artery; such obstruction would almost certainly lead to acute gangrene with perforation.

There is also a developmental factor to be remembered; if any congenital defect exists in the duodenum it invariably occurs at the junction of the second and third stage. Often the second stage ends here blindly—a *cul-de-sac*; in other instances it is greatly stenosed, or joined to the third stage by a fibrous (impervious) cord.

The first and second stages, with the buds which develop into the liver and pancreas and their ducts, appear to be more intimately connected with the stomach than with the third stage of the (adult) duodenum, hence possibly the jejunum begins at the junction of second and third stage of the duodenum, not at the duodeno-jejunal flexure.

It may consequently be normal for delay to occur in the first and second stage, while the passage of the food through the third and fourth stages is at the same rate as food passes along the jejunum.

Even with Jordan's full meal and 4 oz. of bismuth, such is the rate of progress along the jejunum that the food is too distributed to throw a shadow; hence it is possible that, as Jordan's pictures suggest, the food is propelled so quickly along the third and fourth stages of the duodenum as to be invisible.

All the cases whose duodenums were shown (X ray) by Dr. Jordan were those of females, and thin females. Yet duodenal ulcers are found more commonly in men than in women. Why is it that intestinal stasis, *the cause* of duodenal ulcers according to Mr. Lane, is so infrequently seen in men? And, conversely, why the frequence of stasis in women, yet the greater relative frequence of gastric ulcer in women?

Hardly a duodenum shown last week on the screen is abnormal in relation to the adjacent vertebræ, but is normally opposite the first to third lumbar vertebræ. Furthermore, what evidence of dilatation have we been shown? The duodenum is normally wider, *i. e.* of greater diameter, than any other part of the small intestine, and, with the exception of the cæcum and adjacent colon and ampulla of the rectum, is greater than the normal colon.

I see in all reprints of Mr. Jordan's papers the same figures are reproduced time after time.

The case for chronic intestinal stasis is therefore far from proved.

February 17th, 1913.

* Wilkie, 'Path. Dept. Edin. Univ.'

CHRONIC INTESTINAL STASIS.*

(*Discussion continued from p.* 304.)

Dr. E. G. SCHLESINGER said that the alimentary canal as a primary focus of disease had a far-reaching importance which was only just beginning to be recognised. That the evolutionary bands controlling the intestine, which Mr. Arbuthnot Lane had so clearly described, actually existed was now admitted by everyone, and their pathological importance was recognised very generally, excepting by those who from lack of interest or opportunity had not studied the question. That a certain group of symptoms was produced when the development of these bands had passed the limits of physiological utility had also ceased to be a matter for argument, and their interest centred now around the methods of production of these symptoms and the indications for, and benefits of surgery in, their treatment. As Mr. Arbuthnot Lane's house-surgeon he had had ample opportunity of observing these patients before, at, and after operation, and had been able to arrive at certain very definite conclusions on the subject. The clinical picture of intestinal stasis was becoming fairly generally recognised in its more exaggerated forms, but the early cases, those in which, if properly treated, a perfect and permanent cure could be guaranteed, were still to be found labelled as "indigestion," "neurasthenia," "duodenal ulcer," etc., a misery to themselves and a sourse of constant worry and continual disappointment to their medical attendant. The failure to recognise these less obvious cases lay mainly in the fact that constipation was still looked on as synonymous with intestinal stasis. In reality constipation in itself, apart from the mechanical alterations it helped to bring about, was of no importance whatever, and was merely a fairly common symptom in the clinical condition under discussion, whilst people who were constipated were not necessarily the subjects of intestinal stasis. Cases of intestinal stasis fell more or less naturally into certain clinical groups which might be termed the *obstructive*, the *toxic* or *supra-renal*, the *mixed*, and the *end-result* groups, according to the symptoms which predominated. The obstructive type was usually regarded as having a

* At the Medical Society of London.

duodenal ulcer, a gastric growth, or what was known as "nervous dyspepsia." The patient, more usually a man, complained of pain after food, sometimes at once, sometimes after an interval, with vomiting, which usually relieved the pain. The vomit consisted of the food which had lately been eaten, and was quite different from the vomiting which occurred in the second class of case. Headache, cold hands and feet, pigmentation, and the usual "toxic" symptoms might be present, but were often not very marked, and it was not for these that the patient sought advice. These patients were usually the possessors of thick arteries and had a raised blood-pressure. They usually had quite well-developed abdominal muscles. On examination they were very tender over the duodenum and pointed to this as the site of their pain. They might or might not complain of pain in the iliac fossa, but were invariably extremely tender on pressure in this region, and the end of their ileum was readily palpated as a thick tender cord. An intelligent X-ray examination showed a firmly fixed point at the ileum, and a dilated and hypertrophied duodenum. The effects of ileo-colostomy on this type of case were most striking. The patient could eat and enjoy solid food when he had often taken nothing but fluids for months; his blood-pressure fell in a few days to near normal, and he could do the hardest and most sustained work with impunity. It was in this group of cases that paraffin and other non-operative measures were worthless, whilst the operative results were the best, because the ileal and duodenal obstruction was the main feature, and brought the patient to the surgeon while the condition of toxæmia was yet in its early stages. At the operations on these cases the ileum was found to be hypertrophied proximal to Lane's kink up to five or six times its normal thickness, and the duodenum, which might or might not be ulcerated, was also hypertrophied and distended. Bleeding often took place from this distended duodenum even without any actual ulceration. In all cases of this type that he had followed out, the operation of ileo-colostomy had been followed without exception by a permanent return to complete and perfect health. This class of case would derive considerable temporary benefit from a gastro-enterostomy, which freed the duodenal outlet by undoing the kink that developed at the duodeno-jejunal junction, but the continuance of the ileal obstruction was sufficient to cause an early and inevitable relapse. In the obstructive group might be placed many of the cases of so-called Hirchsprung's disease occurring in adults, and the cases of chronic volvulus of the sigmoid. The second group of cases, the *toxic* or *supra-renal type*, occurred more generally in women, and was the group most commonly recognised In these people constipation was generally present, and, unless it had led to that condition of irritative diarrhœa and paralytic secretion (commonly called mucous colitis), was often extreme. The direct abdominal symptoms, apart from the constipation, were, however, overshadowed by the group of symptoms called toxic, which were usually regarded as being due to the excessive absorption of bacterial toxins, but for some of which he offered another and simpler explanation. These symptoms were both subjective and objective, and were often of such intensity as to make the patient welcome death, since life had become unbearable. Chief among these symptoms was a condition of mental depression and general misery, often bordering on madness, which was only increased as measure after measure that promised relief was tried and the condition grew steadily worse. This type of case showed, as a rule, marked pigmentation, had a low blood-pressure, cold and clammy hands and feet, and exhibited a general asthenia very suggestive of Addison's disease. Vomiting was practically constant, but differed from that met with in the obstructive type of case in that it was not related to food, that usually only a watery fluid was brought up, and that it was associated with a deal of distressing retching. Perspiration was profuse and often foul-smelling, morning headache was constant, the skin was harsh and inelastic, and often showed the development of a fine hairy down. Cystic degeneration of the breast was frequent and might later take on the characters of malignant disease. Pyorrhœa alveolaris was often extensive, and added not a little to the wretched condition in which these people were. In typical cases of this group the patient might not complain of any abdominal pain, but on examination there was tenderness over the end of the ileum and over the sigmoid, which was straight and readily palpated. The bands which straightened out the sigmoid might implicate the left ovary. The general condition of the patient was often regarded as due to this ovarian

condition; the ovary was frequently removed in consequence, while occasionally the right ovary was similarly implicated, and a double oöphorectomy with its concomitant miseries was added to the patient's other sufferings. X-ray examination in these cases might fail to detect an ileal kink, and, indeed, it might be found to be absent on operation, the patient's natural reactive tendency being in abeyance. This was the extreme picture of this type of case, but all stages of the condition, from the patient who had occasional "bilious attacks," through the various types of indigestion, atonic constipation, neurosis, etc., could be recognised. This group of cases ought never to need the operation of ileo colostomy, since, if the correct nature of the condition was recognised sufficiently early, liquid paraffin, an abdominal support, and a healthy married life would work wonders. Unfortunately, however, this was the type of patient on whom medicines, diets, spa treatments and operations were showered, and the underlying cause of the condition was not recognised until conservative treatment was out of the question, and until the general condition of the patient and the rottenness of the intestine made the operation of ileo-colostomy a doubly difficult one, and rendered the return of the patient to *complete* health uncertain. The combination of asthenia, pigmentation, low blood-pressure and vomiting which was to be found in cases of this type was so suggestive of the clinical syndrome met with in Addison's disease, that one had been struck by the possibility of one underlying factor being capable of explaining the two conditions. Whatever might be the true pathology of Addison's disease, there could be no doubt that a deficiency of adrenalin was, at any rate, an important factor, and that this deficiency was brought about by a destruction of the suprarenal gland by tuberculosis or other means. It seemed, however, plain that there was another manner in which the body could become short of adrenalin, or for that matter of any internal secretion, and that was by a shortage of the precursor in the alimentary tract from which the internal secretion was metabolised. That in the human body the various amino-acids which resulted from the breakdown of proteins were used as a basis for the manufacture of the various more complex substances met with seemed to be definitely proved by the many feeding experiments which had been carried out. The human body was incapable of building up its more complex constituents from their ultimate breakdown products, and had to start with the stage amino-acid. These various amino-acids seemed to subserve a very definite and specific function, and in all cases reached the body by way of the alimentary canal. It seemed, therefore, possible that if these amino-acids were in some way altered in the alimentary canal so that their absorption as such was deficient, there would be a corresponding deficiency of that substance for which they served as the basis. In other words, a deficiency of any internal secretion could result in two distinct ways: either from destruction of the organ whose function it was to produce that secretion, or from a deficiency of the precursor of the secretion in question, without which the body was unable to manufacture it. It was this latter theory which, he held, explained the deficiency which analogy led him to believe was present in the "toxic" group of patients with intestinal stasis. The important factor in this group was not so much the actual delay in the passage of material through the intestine as the infection of the small intestine which resulted from the delay. Dr. J. W. H. Eyre had kindly investigated swabs taken from the ileum at the time of operation on several of these cases, and had obtained in each case a copious growth, mostly of coliform bacilli, but in one case of a pure *Streptococcus longus*. What, then, was the effect of this excessive growth of organisms in the small intestine? Principally that bacterial digestion, which normally took place in the poorly absorptive colon, now occurred in the ileum. There was still some doubt as to the amino-acid from which adrenalin was built up, but the chemical constitution of tyrosin and adrenalin was sufficiently close to be very suggestive, and the transformation of the one to the other by the suprarenal gland had a certain amount of support from experiments which had been carried out on the subject. It was known that the action of bacteria on tyrosin resulted in its splitting up, and he would apply this fact to explain some of the symptoms of intestinal stasis. The abnormal presence of bacteria in the small intestine resulted in a decomposition of the tyrosin, which was therefore absorbed in deficient amount, and consequently the suprarenal gland, being supplied with a deficiency of the precursor of adrenalin, was able

to manufacture only a deficient amount, and a deficiency of adrenalin proportionate to the amount of intestinal infection with the corresponding symptoms resulted. The excessive pigmentation which occurred in these patients was capable of a similar explanation. The melanins on decomposition yielded a large amount of indol, and it was the view of many pathologists that they were built up from indol, and that the sulphur and iron were added in the tissues. Tryptophane, an amino-acid resulting from protein digestion, was further broken down by bacteria into indol and skatol. Consequently, in the infection of the small intestine which occurred in intestinal stasis there was an abnormal quantity of indol and skatol produced, which, when absorbed slowly, was very probably metabolised to a melanin and deposited in the tissues as such. The fact remained that after an ileo-colostomy, which, by adequately draining the small intestine, presumably freed it rapidly from its infection, the disappearance of the pigmentation was one of the speediest and mort striking results. The theory that alterations in the various internal secretions were not of necessity dependent on lesions of the organs which produced them had a wide application, and when physiological chemistry was able to tell them more about the composition and origin of the internal secretions, it might be able to explain some of the many interesting problems connected with them. All that they would maintain at present was that an internal secretion did not begin and end with the organ that produced it, but that an adequate supply of the precursor from which it was built up was of equal importance. Cases of this toxic or suprarenal group were greatly improved after an ileo-colostomy, the blood-pressure rose to near the normal, the pigmentation and coldness of the hands and feet disappeared, and the general listlessness and lack of energy were greatly improved. Nevertheless, these patients were operated on so late that many of the changes, more particularly the mental changes, had become permanent, and the patient, although almost always much bettter, did not return to complete health in the way that patients of the first type did. When the history of these cases was examined this was not surprising, since their illness often extended over many years, and they only reached the surgeon when everything else had been tried and had failed. The *mixed* group of cases included those patients who presented a mixture of what he had called the "obstructive" with what he had called the "toxic" symptoms. In these cases the more marked the obstructive symptoms were, as opposed to the toxic, the better were the results after an ileo-colostomy. The least satisfactory of all were those patients who had a well-marked ileal stasis, and who yet, on operation, showed no trace of an ileal kink. These were the patients to whom Mr. Lane applied the term "simple stasis." The reason that cases with well-marked "obstructive" symptoms did better than the others appeared to be simply that the severe abdominal pain in the obstructive type led them to seek surgical aid much earlier than the patients whose chief complaint was the misery of "toxæmia," and consequently their intestine was better able to adapt itself to the new conditions and to carry out its functions when the obstructing bands had been put out of action. The group to which the term "*end-result*" was applied was a large and varied one, and consisted of cases in whom the end-result had reached such proportions as to overshadow the underlying cause. This group included cases of such diverse nature as trigeminal neuralgia, many cases of dementia, of all stones, pancreatitis, many cases of cancer of the breast, etc. It also included a large group of patients in whom the general resistance had been so lowered as the result of intestinal stasis that the body was a ready prey to various infective processes. Under this head came many cases of tuberculosis, rheumatoid arthritis, Still's disease, etc. In this last group it was often a nice question whether the end-result had not become too advanced for it to be worth while to deal with the stasis, and, of course, in the case of malignant disease this was out of the question. It was this group of cases whose boundary was being daily extended, and when as much attention was paid to a systematic examination of the alimentary canal as was at present given to auscultation and percussion of the chest, there could be little doubt that many conditions at present looked on as primary would be found to be in the first instance dependent upon abnormalities in the mechanics, physiology, or bacteriology of the alimentary canal.

Mr. L. E. CREASY said that he had had an opportunity of examining these cases before, during, and after operation. The symptoms which the patients chiefly complained of were indigestion, impeded respiration, tenderness over the cæcum, pain below the last rib on the right side, pain in the loin, dull pain over the right sacro-iliac joint, griping pain, and severe pain during the passage of fæces. The left ovary was often tender. Other symptoms were mental depression, faintness, neuralgia, irritability, loss of fat, and staining and darkening of the skin. He could say from close observation that the treatment advocated by Mr. Lane abolished these symptoms.

Dr. DISTASO said that constipation was a transitory or chronic condition of toxæmia which arose from the fact that toxins were liberated in the intestinal content owing to prolonged stasis. This condition produced a change in the intestinal flora in which an almost entire disappearance was observed of certain microbes which were usually present in the cæcum. The absorption of those products by the organism caused intoxication of the human body, whose powers of resistance were

thereby diminished. In order to support his statements he must show that constipated persons were intoxicated ; further, he must show how and where the toxins were produced, and finally, that by changing the intestinal flora the intoxication disappeared. He would also try to define the transitory or chronic condition in constipation—an important matter which would lead them to consider its evolution, since the transitory state might become chronic. As to whether a constipated person was intoxicated he would waste no words and time in discussing, as all knew well the particular tint and colour of the skin, the expression of the eyes, and the dull look and appearance. The subjective symptoms were also familiar—namely, the phenomena affecting the brain and morals. All these subjective and objective symptoms were bound up with the intestinal stasis. There was therefore no doubt that they were related as cause and effect. Mr. Lane, in his papers on this subject, gave a clinical tableau of the matter. He could not add anything clearer and more thorough than what Mr. Lane had observed. He would discuss the second question, which was, in his opinion, a most important one—namely, the seat of the intoxication. If in constipation the large bowel were removed and the intoxication was suppressed, one might conclude that the cause of constipation resided in the large bowel. We owed, again, to Mr. Lane the demonstration of this fundamental principle on which the theory of constipation and intoxication was based. Mr. Lane removed the large bowel from a constipated patient, and the patient was cured both from constipation and intoxication. What phenomena took place in the digestive tract of a constipated person? He had nothing to add to the different papers read at the last meeting and illustrated by radiograms. He would only try to sum up the observations made, and look upon them from a synthetic point of view. The first statement which supported the opinion previously mentioned was, that all authors admitted with remarkable accord that the phenomenon of constipation took place in the cæcum and in a small part of the ascending colon ; secondly, the formation of the haustra in the ascending colon ; thirdly, the formation of lumps in these parts ; fourthly, the existence of the peristalic waves ; fifthly, the stasis can proceed so far as to occur in the lower part of the ileum, and consequently dry fæces result. With regard to the first three points, he need only quote the comprehensive works on "radiography," by Hertz, Schwarz,* Stierlin,† and Albrecht.‡ With regard to the fourth point the anti-peristaltic movements of the intestine in constipation were also an undisputed fact. Though they might have escaped attention, they nevertheless occurred also in normal persons. He would only refer to physiology, and more especially to the splendid book just published by Cannon. He would like, however, to point out a truth which seemed to him to be of the utmost interest—namely, the existence of ant -peristaltic waves in normal animals. Cannon showed that they were present in cats under normal conditions. Elliot and Barkley Smith assumed that in man the material entering the proximal colon "was still delayed by a backward current, still commingled by the activity of the walls of the sacculi." There was also no doubt that they existed in normal man, but they were too difficult to observe. On the other hand, he must point out a fact of daily observation which led him to admit the existence of these antiperistaltic waves in normal man. It was known that if one had the *stimulus defecatio* and neglected to answer it one become constipated, or was in danger of becoming so. If one did not admit that anti-peristaltic movements took place, then one must admit retrograde movements. This idea was gaining more ground, and he had just lately seen a radiogram by Schwarz, who pointed out that in some cases of constipation the *globus pelvicus* went back to the cæcum. Schwarz* called the condition *dyschinetische obstipation*, and gave some observations by Rieder, who found similar movements in normal persons. This was important, but their existence in constipated people was far more important. He did not believe that the existence of these movements would any longer be denied. He would shortly discuss their effect. They converted the cæcum into a veritable shaking apparatus where the fæces, through going backwards and forwards, became dry ; and as the movements were accompanied with an enormous production of gas,† they caused the cæcum to swell, the walls to be distended, and through their becoming thinner, the bacterial products easily passed through. They resembled a filter which had been used for a long time. Consequently the walls ceased to act in a normal manner, and the strength of the peristaltic waves was diminished. As a consequence of the stasis one might easily imagine what were the results upon the entire digestive system. These were well illustrated by Dr. Jordan's radiograms, who, in accord with Mr. Lane, showed the dropping of the viscera. Such was the state of affairs in chronic constipation, which doubtless arose owing to the condition of the colon, which did not work properly, and so prevented the passing on of the remains of digestion in the cæcum. Therefore the power of regulation of the descent of the remnants of digestion was lost. The consequence was that stasis began in the upper part of the intestine with the dropping of the viscera, the formation of the

* 'Munch. med. Woch.,' 1911–1912.
† *Ibid.*, 1912.
‡ *Ibid.*, 1912.

* *Ibid.*, No. 40, 1912.
† Shown by radiograph, G. Stierlin, 'Munch. med. Woch.,' No. 36, 1911.

kink, and the absorption from the small intestine of the toxic substances produced. These conditions were the worst owing to the fact that the upper part of the intestine absorbed the decomposition products with greater facility. These conditions, as observed by radiograms, resembled those found in guinea-pigs and rabbits, which were both typically constipated. If a guinea-pig's abdomen was opened up, one was struck by the fact that there was a marked agreement with the radiograms. He would consider, after having discussed the mechanical facts which accompany constipation, what took place inside the intestines. The fæces underwent certain changes. There was no doubt that the cæcum of constipated persons contained the same microbes which were present in normal persons. He confirmed this fact by examining the cæcum of three constipated people. If the fæces of constipated people were examined two things in particular would be observed. First, the fæces resembled those of a goat; they smelt very badly, had a characteristic odour, an acid reaction, and were almost black and dry. In other people, especially in the old, the fæces were of quite a different aspect. To understand what took place in the intestinal flora of a constipated man, one must consider first the composition of a normal man's intestinal flora. A counter-stained film showed the presence of Gram-negative microbes belonging to the *coli* group, which were predominant, and also an enormous number of microbes generally. A similarly stained film from a constipated person showed an extraordinary diminution of the amount of microbes; secondly, the Gram-negative microbes which were usually predominant had almost entirely disappeared. Gram-positive microbes were now in the majority, and only a few microbes were present. From this he assumed that the microbes had disappeared in the large bowel, and had in all probability disappeared in the cæcum, where radiograms had shown that the fæces could remain for sixty hours or more. In constipated persons the *coli* group microbes were broken down, and therefore the so-called endotoxins became free and produced inflammation of the colon with resulting intoxication. One must remember the well-established fact in bacteriology that that group of microbes was especially known for giving the so-called endotoxin, the poison which was included in the bacterial body and which required its breaking down to become free. Before making experiments he was a believer in the view of intoxication through substances of the aromatic series, but now he was of opinion that in constipation the intoxication was principally due to the endotoxin which became free. He would not deny that indol was able to produce sclerosis of the organs, but that was not the principal cause, as the poisoning through the liberated endotoxin of the microbes belonging to the *coli* group was very severe, and only this could explain the appearance of the organisms in constipation which seemed to be an intoxication, like that of cholera or dysentery. This suggestion, too, was based upon well-established observations. He had a laboratory attendant, a tall and healthy man, whose fæces were soft, abundant, and acid, and who did not remember ever having been constipated. Dr. Distaso had examined the man's fæces for almost two years, and his urine also. The amount of indican in his urine was enormous; there was a true indicanuria. On the other hand, the urine of six constipated persons, examined by Mr. Garratt, showed only in one case a slight trace of indican. If that was the measure of the intoxication of the body through the micro-organism of the fæces, those persons, accord- to that theory, were not at all intoxicated. The second point, that of the clinical picture in constipation, led him also to think that the symptoms observed were due to a toxæmia of bacterial origin. Constipated people suffered from headache, cold hands, cachexia, often a slight rise of temperature, and nervous and circulatory disturbances. These symptoms suggested a toxic action of bacterial origin. Of the utmost importance was the relation between constipation and nervous diseases. A medical friend of his was good enough to communicate to him the following observation: A young man came to him who had many attacks of epilepsy every day; he thought the constipation might be the cause of his disease. He cured the constipation and the man's trouble too. The man was now on the stage and earned a good deal of money, and after a wretched existence was now enjoying his life. Obviously the relationship between the two phenomena was one of cause and effect. He would refer to another group of phenomena in which constipation played an important *rôle*. The improvement in tuberculous children when Mr. Lane removed the large bowel had been mentioned. They had also heard of a case which had been sent to the hospital for cancer of the breast, which after short-circuiting became almost normal. He could mention a case from Mr. Lane's clinic of well-developed goitre. He had seen the reduction of the goitre till it became of insignificant proportions after the short-circuiting. Of course, the primary cause remained still, but intoxication through the microbes of the large bowel had had the power to make worse the primary infectious process. He would classify these cases under the name of *intoxication surajoutée*, where the intoxication acted like the symbiosis of the infectious diseases. He spoke shortly about the relation between diarrhœa and constipation. Both, as he had stated in previous papers, were aspects of the same phenomenon. Dr. Jordan had mentioned a case of muco-membranous colitis, in which the radiogram showed a condition of

constipation of the upper part of the intestine. How could one explain the presence of both constipation and diarrhœa? The answer was to be found in the fact that toxins in the general circulation acted upon the distal part of the intestine and produced diarrhœa with the constipation. As a matter of fact, this hypothesis was founded on other data. The diarrhœa of tuberculous colitis doubtless in the first stage of its existence was caused through the circulating poison, because only in the second stage was ulceration found. He would not deny that in some cases of colitis they had to deal with specific microbes, as he had pointed out in a summary on the bacteriology of colitis ('Soc. Biol.,' 1912). Another matter of great importance was the bacteriological findings both in constipation and diarrhœa. If one made a Drigalski plate from cases of diarrhœa not due to specific microbes, one saw that the *B. coli* of Escherich had almost disappeared, whilst other microbes were present. In constipation this was also true. These facts established a point of resemblance between diseases with opposite symptoms, but most probably due to the same causes. It was also a fact that the goat and the cow had the same anatomical and physiological features of the intestine, but the cow had diarrhœa, whilst the goat was a typically constipated animal. This could not be explained without admitting a common cause of both troubles. He considered briefly the last part of his statement as applied *in vivo*. If constipation was really a disease, owing to a special change of the intestinal flora it would be possible to produce such changes experimentally in an animal or in man. He chose a dog because it ate anything, and he could make variations in his experiments. The dog normally had hard fæces, and it would be, therefore, unsuitable for experiments under such conditions. In order to overcome this difficulty he put the dog on a vegetarian diet. The fæces became at once very soft. The flora was completely changed, and the predominance of Gram-negative microbes became evident. He fed the dog for some time on this diet, and afterwards added 15 to 20 grm. of meat. The external appearance of the fæces changed immediately. Gram-positive microbes were in predominance in the intestinal flora and the fæces became hard, like those in constipated persons. Monkeys showed nearly the same changes. He made further experiments on the subject to show how the food and the intestinal flora had an influence upon the large bowel and on constipation. Guinea-pigs fed with cabbage were typically constipated. If a guinea-pig a day old was given no cabbage at all, but peas and milk, it had always soft fæces and a different intestinal flora. A big dinner, for instance, had often this effect. He would quote two facts in his experience which, he believed, were instructive. He went from Italy to Switzerland and became at once strongly constipated. He came from France over to England and again became constipated. As he stated in his previous paper, this connection between the remnants of digestion and the intestinal flora made the condition very serious. He called this state "labile condition," which, like unstable equilibrium, might very easily be upset. These experiments point out clearly that: (1) The intestinal flora was an outcome of the kind of food; and (2) the products of the digestion which descended into the cæcum were used by the microbes which produced certain substances inhibiting the stimulation of the intestine. This second point was important, because it afforded an explanation of the way in which constipation took place. With 15 grm. of meat the intestinal flora of the dog was changed and the animal became constipated. What was the explanation of this? It had been seen previously that the microbes of the cæcum were the same in normal and constipated man, and that the remnants of the digestion could remain in the cæcum sixty hours and more. They knew, on the other hand, that the microbes in the cæcum were capable of producing different substances with different material. He would quote an example which explained this. If one took *Bacillus coli*, or an allied microbe, and placed it in sugar media, there was a notable production of acid. On the contrary, if the same *Bacillus coli* was placed in the presence of decomposed albumen it produced very offensively smelling products. Just the same happened in his experiment. A little meat had caused the descent into the cæcum of some decomposed albumen which was utilised by the *coli* and other organisms, and therefore such substances as were able to lead to intestinal stasis were produced. This was, in his opinion, the primary cause of constipation. A fault in the diet provoked such a state, at first transitory, later chronic, and he would strongly point out that perhaps constipation in the adult was the consequence of mistakes in childhood. If constipation was a disease due to the intestinal flora, they should be able to cure it through the changing of the flora. He would give his results without pretending to have settled the question. The cases he had cured numbered three. (1) A Finnish doctor who was believed to suffer from cancer. He had been able to change his flora and obtain a cure. (2) A Danish girl, æt. 23 years, who suffered severely from constipation. All treatment had done her no good. He was fortunate enough to obtain her stool daily. (3) An English lady, æt. 25 years, who got well, but was lost sight of. He thanked them for having willingly undergone his experiments. These were his results, but he was sorry not to be a practitioner to prove if his method was of any use or not.

(*To be concluded.*)

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ECZEMA, AND SOME ERUPTIONS OFTEN MISTAKEN FOR ECZEMA.

By H. G. ADAMSON, M.D., F.R.C.P.,

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ECZEMA was at one time regarded as the most common of all skin diseases. Of recent years its field has become gradually more and more limited, until, for some observers, the disease, eczema, threatens to disappear entirely. But this, in the opinion of others, is by no means the case. What is happening is that many eruptions, which were formerly included under this head, are now put outside the eczema group, and eczema itself is becoming more clearly defined as a disease with very special characters.

It is now generally recognised that eczema is essentially an affection of non-microbic origin, and for this, as well as for other reasons, the following eruptions must be separated from the eczemas: Impetigo contagiosa of streptococcic origin, and impetigo of staphylococcic origin, including many chronic forms of dermatitis probably due to one or both of these organisms; the so-called seborrhœic dermatitis, which is probably of microbic origin and related to the impetigoes; certain chronic eczematoid eruptions of the fingers and toes due to ringworm fungus.

There are also two other eruptions of non-microbic origin, which, although having some characters in common with eczema, are to be excluded from that group, namely cheiropompholyx and lichen chronicus simplex of Vidal or lichenification.

Having to some extent cleared the way by recalling these eruptions, I shall now proceed to the consideration of eczema itself.

DEFINITION.

I have already said that eczema is to be regarded as a very distinct disease, with very definite

characters, and I would define it as a *catarrhal inflammation* of the skin of non-microbic origin, resulting from some local external irritation in a predisposed person.

I shall now deal with these characters more in detail:

(*a*) *Catarrhal inflammation.*—By catarrhal inflammation we mean an inflammation in which serous exudation is the prominent feature rather than cell exudation. If we examine microscopically a section of an eczema eruption we find that everything points to inflammatory congestion with œdema of the tissues; the blood-vessels in the corium are dilated, and the prickle-cells of the epidermis are swollen and separated by serum in places to form intra-dermic vesicles. Clinically, corresponding with these histological features, we see more or less sharply circumscribed areas of skin which are reddened, swollen, and covered with minute vesicles or weeping points.

The non-microbic origin of eczema.—For many years there have been keen discussions as to whether eczema is or is not of microbic origin. The tendency is more and more to regard eczema as a non-microbic eruption; and any eruption which is proved to be of microbic origin is to be removed from the eczema group. The primary eczema vesicle has been shown to be amicrobic. Cocci, with staphylococci and streptococci, are found in lesions of less recent origin, and there is no doubt that eczema may be complicated by staphylococcic and by streptococcic infections, and perhaps aggravated and kept up by the toxins of these organisms; but these are secondary infections, and the underlying eczema is not primarily due to their invasion.

External irritants and predisposing causes.—Anyone who has the opportunity of studying closely and of treating many cases of eczema must be convinced of the importance of local irritation as a factor in the production and maintenance of the eruption. That there is some predisposing condition which renders the patient's skin very susceptible to local irritants there can also be no doubt. But the more one studies these cases, the more difficult it becomes to fix upon any particular predisposing cause.

An illustration may be found in that very common form of eczema which is known as trade or occupation eczema. Persons engaged in occupations which necessitate exposure of the hands to irritating dusts or fluids—barmen, washerwomen, bricklayers, packers, French polishers, charwomen, photographers, museum attendants who handle formalin specimens, and many others—are liable to eczema of the hands. A very striking thing about these cases is that the patient may have previously followed the same occupation for years without having eczema. It is evident that a hypersensitiveness to local irritants has developed: what is the nature of the predisposing influence we do not know; but it is obvious that local irritation has much to do with the appearance and continuance of the eruption, for we find that it gets better on stopping the work and bad again when the work is resumed.

In all cases of eczema we find a great susceptibility to local irritants, so that frequent washing, exposure to heat and cold, slight frictions, etc., which in a normal skin would produce no reaction, in these cases result in an eczematous eruption or aggravate an existing one. It seems probable that these patients are made susceptible by some toxin, the result of some metabolic disturbance, but we have no clue as to its nature. It has for long been the custom to speak of gouty eczema, but all we can say upon this point is that some patients who suffer from eczema belong to what is generally called a lithæmic type—a predisposition in the individual or his family to gout, gravel, migraine, asthma, bronchitis, diabetes and eczema. The French use the term "arthritic" to express the same meaning. But it is by no means clear that all persons who suffer from eczema are of lithæmic tendency.

Clinical Features of Eczema.

We will now turn to the clinical side. It has been customary to classify eczemas according to (1) lesional varieties; (2) regional varieties. Amongst lesional varieties there are described Eczema erythematosum, E. vesiculosum, E. pustulosum, E. rubrum or madidans, E. squamosum, E. rimosum. Some of these, as E. pustulosum, are no longer regarded as eczemas. The majority merely describe some one feature which is more prominent in that particular case, but which is common to all eczemas.

In the characters already mentioned, of redness and swelling of the skin with pin-point vesicles

over the red, swollen surface, together with the symptoms of burning or itching, and the circumstance that the eruption is aggravated by local irritation and calmed by protection from such iritation, we have a description which applies to every case of eczema. One of the most striking of these features, but one which is often overlooked, is that of œdema of the skin in the affected area—a feature which is apparent to the sight, but which is more readily appreciated by pinching up the skin between the finger and thumb, when it will be found to be twice or three times as thick as the normal skin. As secondary results of this vesication in the œdematous areas we may get crusting (eczema crustosum); or the minute broken vesicles may be so closely set as to form raw surfaces (eczema madidans); or in parts where there is constant movement the œdematous areas may become deeply fissured (eczema rimosum).

Then in regard to the division into regional varieties, E. capitis, E. faciei, E. palmaris, etc., we find also that many of these old terms apply to diseases which are not now regarded as eczema.

So that for practical purposes it is more convenient to classify eczemas according to certain clinical types, rather than according to the appearance of the lesions or their distribution. The main clinical types are as follows :

(1) Trade eczemas or occupation eczemas.
(2) Nummular or patchy eczemas.
(3) Erythematous eczemas.
(4) Eczema of the palms and soles.
(5) Infantile eczema.

Trade eczemas or occupation eczemas.—The eruption occurs about the fingers, hands or forearms of those whose occupation involves contact with local irritants. Eczema of this type may vary in severity from a few red, swollen patches covered with pin-point vesicles, up to involvement of the greater part of the skin of the hands, fingers, and forearms. The photograph (Fig. 1) is that of a mild case in a washerwoman, and here the groups of minute vesicles are clearly seen—the redness of the patches does not, of course, show in the photograph. On pinching up the skin it was thickened by œdema.

In a case of trade eczema, the eruption may eventually spread to other parts of the limbs as nummular eczema, and to the face as erythematous eczema.

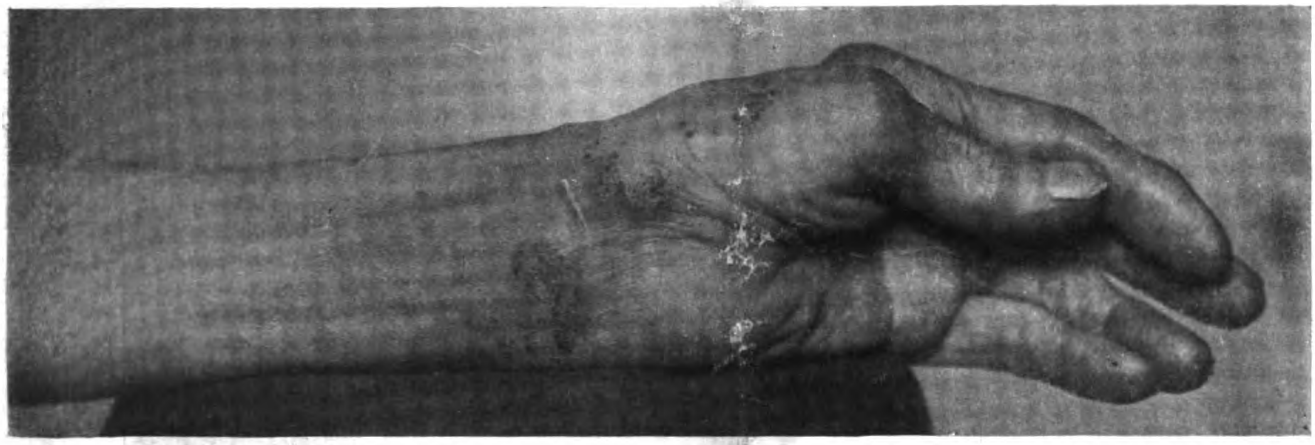

FIG. 1.—Trade eczema. Typical patches of eczema—red, swollen, and covered with minute vesicles or erosions.

Nummular or patchy eczema.—So called because the eczema occurs in patches, the patches being most abundant generally upon the arms and legs. Cases of this sort, in which the patient presents nummular patches of eczema upon the arms and legs, may occur as such from the beginning, or they may be, as has just been stated, secondary to trade eczemas. In this photograph (Fig. 2) the patches present all the characters I have mentioned; they are more or less circumscribed, they are red, the skin is œdematous, so that when pinched up the fold is of two or three times the thickness of the normal skin. They are covered with pin-point vesicles, or small crusts, or the whole surface may be more or less excoriated and crusted. They itch intensely, and a characteristic feature is that they are made worse by scratching or other local irritation, and subside when protected from such irritation.

Erythematous eczema is the term applied to

eczema when it occurs on the face in adults, or on the sacrum and penis. In these situations, on account of the looseness of the cellular tissue, there often occurs a considerable swelling of the face, especially about the eyelids, and of the scrotum and penis. The redness and swelling may be so marked that such cases may be mistaken for erysipelas, though there is generally here and there sufficient evidence of pin-point vesication to point to the true nature of the case. Erythematous eczema generally forms parts of a more generalised eczema, which include the hands, and often also nummular patches on the arms and legs.

Chronic eczema.—This term may be applied to any case of eczema which continues over a long period. Essentially the features are the same—in fact, we have to do with a series of acute or sub-acute outbursts. But in long-standing cases there may be an œdematous thickening of the skin, which is present even between the acute outbursts.

Palmar eczemas are also common, the eczema being confined to the palm, or, as is often the case, involving also the backs of the fingers. Such cases are often difficult to distinguish from syphilis or psoriasis of the palms. They are distinguished by the fact that there is itching, and by the discovery of minute vesicles beneath the epidermis at some part of the eruption, and sometimes by the fact that there is eczema on the backs of the fingers or elsewhere (Fig. 3).

Infantile eczema.—The eruption of eczema in babies has all the features of eczema in older subjects, but its special characteristic is its peculiar distribution. It occurs commonly between the ages of six weeks and two years. It is always present upon the face; in mild cases it attacks the cheeks and forehead, in severe cases it extends over the whole face with the exception of the skin around the eyes, nose and mouth. Sometimes the arms and legs may be involved, but never without the face.

Fig. 2.—Patchy eczema of the forearm. The red swollen areas have been eroded by the dressing, which had been allowed to "stick."

The peculiar mask-like distribution on the face is well seen in Fig. 4. In mild cases the eruption may be little more than a redness, with thickness from œdema, of the skin of the cheeks, with a pin-point vesication of the œdematous area. In more severe cases there may be oozing with heaping up of crusts. In the majority of cases eczema of babies disappears before the age of two years, but in severe cases, if not efficiently treated,

it may go on into childhood and then it loses its characteristic distribution on the face and becomes a patchy eczema of the limbs and trunk.

A not uncommon situation of the eczema patches in these chronic cases in children is behind the knees.

Such cases of chronic eczema in infants and children may continue into adult life unless means be taken to prevent the constant scratching which keeps up the eruption.

Diagnosis.

In diagnosing eczema from other skin eruptions, the most important points to remember are: that in eczema we have an eruption which itches, which is made worse by scratching, and which subsides when protected; that it is a catarrhal inflammation, so that we find redness, with œdema of the skin (which is evident on pinching up the skin); and that the red œdematous area is studded with pin-point vesicles, crusts, or erosions.

Probably the affection most often mistaken for eczema is scabies, and in any case of suspected eczema the possibility of scabies being present should pass through the mind, and the wrists and fingers should be examined for the burrows of the acarus. In many cases scabies can be at once excluded, but some care may be required when we are dealing with an eczema which attacks the limbs or trunk. It is not uncommon for a case of scabies to be treated for too long a period with sulphur ointment, with the result that there is produced a generalised sulphur dermatitis, which simulates eczema. The only way to avoid falling into the error of mistaking scabies for eczema is to become familiar with the burrow of scabies and with the art of finding the acarus.

Most of the other eruptions which present any difficulty in diagnosis from eczema belong to those which I mentioned at the beginning of this lecture as now being separated from the eczema group, and the diagnosis will be considered when I come presently to give a brief description of them.

Fig. 3.—Palmar eczema.

Treatment of Eczema.

The treatment of eczema may be summed up in three words: protection from irritation. Although we know that in persons who suffer from eczema there is some predisposing factor which renders the skin highly susceptible to local irritations, and we also know that a change of diet, a change of climate, or other alteration in the conditions of life may sometimes lead to the spontaneous disappearance of eczema, yet in the large majority of cases of eczema we fail to find anything which points to an internal cause, and treatment in the form of dieting is altogether ineffectual.

In a few cases, which we may perhaps, in deference to the patient's own views, call "gouty cases," it is an advantage to restrict the meat diet and alcohol. In many cases, however, we find that the patient has been starving by treating himself on anti-gout lines, and needs feeding up. Occasionally one meets with cases in children where eczema is said to alternate with asthma, but although I have several times met with this association, I do not regard it as a common one. In most cases, I repeat, we find no definite predisposing cause, and we do far better to concentrate our attention upon the removal or prevention of local causes.

FIG. 4.—Infantile eczema. Typical mask-like distribution on forehead, cheeks, and chin, the nose, eyes, and mouth remaining free.

Apart from the local irritants of various trades or occupations, eczema may be kept up by strong soaps, the intermittent use of water in the ordinary daily ablutions, friction of rough clothing, the irritation of dust from books and papers, and above all by the rubbing and scratching of the patient. It is generally this last which is responsible for the continuance of the eczema, and it is one of the most difficult factors to eliminate.

In acute eczema, it is advisable, if possible, to keep the patient in bed, at any rate until the acute stage has subsided. One of the best and simplest applications for such cases is a continuous dressing of butter-muslin folded four or five times, and soaked in normal saline solution (common salt ʒj to one pint of boiling water). If there is difficulty in keeping the dressings moist they must be covered with gutta-percha tissue and lightly bound on. Wet dressings should only be employed for a few days, and then an ointment, paste or cream containing zinc oxide should be continuously applied in the manner which I shall presently describe.

Here I should like to say a few words in regard to baths in eczema. In most cases it is a good rule to forbid the use of water at all, except in the form of the dressing just described. But in acute or chronic eczemas, which are extensive, and when

FIG. 5.—Impetigo contagiosa showing the "stuck-on" crusts.

the patient is kept in bed superintended by a nurse, daily bran baths are often beneficial. The bran bath is made by soaking in the bath two or three pounds of bran placed in a bag or pillow-case, and swirling it about until the water is thick. The patient is placed in this bath for ten minutes to a quarter of an hour. No soap is used, and immediately on coming out of the bath the skin is mopped with warm dry towels, and the dressing applied to the eczematous patches, after first dabbing them with lead and calamine lotion.

In subacute cases of eczema we may begin the treatment at once by the use of greasy applications. The ordinary zinc ointment of the Pharmacopœia is an excellent application, but the value

of a zinc ointment is much enhanced by the addition of starch powder to form the well-known zinc oxide paste of Lassar: Zinci oxidi ʒij, pulv. amyli ʒij, vaselin ʒss.

The paste is spread upon butter-muslin (which can be bought at a draper's shop for a few pence a yard) and lightly bound on to the parts affected, The dressing should be changed once or twice in twenty-four hours. At each dressing it is important that the old zinc paste should be carefully removed. This should be done, not with water, but by gently wiping it off with a piece of wool soaked in a liquid vaselin (paraffinum liquidum B.P.). The dressing should never be allowed to get dry, and to prevent this happening it may be necessary to occasionally dab the butter-muslin from the outside with liquid vaseline.

If we can but ensure that the skin is efficiently protected in this way, most eczemas will get well without further treatment. If improvement does not take place, there is some fault in the application of the dressings, or, as is very often the case, the patient is rubbing and scratching the patches.

In some cases which hang fire we may hasten the cure by means of drying-up applications. There are several ways of doing this, of which the best are:

(1) To mop the eczematous patches with lead and calamine lotion at each change of dressing: Liq. plumbi subacet ʒss, glycerine ʒss, calaminæ ʒj, zinci oxidi ʒj, aq. ad. ʒj.

(2) To paint the patches every other day with a solution of silver nitrate: Argent nitratis gr. x–xx, spirit ætheris nitrosi ʒj.

(3) To add to the zinc oxide paste, lenigallol in the proportion of ʒss–ʒj to ʒj.

The effect of the lenigallol (a derivative of pyrogallic acid) is to dry up the wet points, which become covered with little black crusts, which later fall off and leave a dry surface.

For patches of eczema in parts where bandaging is difficult, the salve-mull zinci oxide of Beiersdorf and Co., No. 1011, may be employed instead of the zinc paste. This is a paste, spread on muslin, which may be cut into pieces of the desired size and stuck on to the patch.

FIG. 6.—Impetigo contagiosa of the fingers. The superficial erosions with over-hanging epidermis at their margins (see right little finger) are in reality ruptured bullæ. This is a streptococcal infection.

In infants often the greatest difficulty will be experienced in preventing scratching and rubbing, and until this difficulty has been overcome no progress will be made. The zinc paste must be applied on a mask of butter-muslin with holes for the eyes, nose and mouth, and lightly bound on. The child's arms must be put into splints, made by placing a roll of cardboard round the elbow so as to prevent the arms being flexed. The child can then move its arms, but cannot reach its face.

Most eczemas can be cured by careful attention

to the protective measures I have just described, but there are a few further applications which are useful in special circumstances.

In chronic eczema of the palms with much thickening of the epidermis, treatment may be commenced by the addition of salicylic acid gr. xx to ℥j of the zinc paste, using the simple paste after the epidermis has been peeled off. In erythematous eczema of the face in adults, the lead and calamine lotion, already mentioned, may be mopped on several times daily, instead of using ointments. But care must be taken not to allow the powder in the lotion to get caked on, and this is best done by mopping with oatmeal-water before each fresh application of lotion.

For chronic patches of eczema X-ray applications are of great value, and will remove such patches when all other methods have failed.

I shall now very briefly describe those eruptions

FIG. 7.—Sycosis. A follicular pustulation of staphylococcic origin sometimes wrongly called pustular eczema.

which I mentioned earlier as having been separated from eczema, or which are at the present time regarded by many as affections which ought to be separated from eczema.

The first of these eruptions to be clearly distinguished from eczema was *impetigo contagiosa*, first described as a distinct disease by Tilbury Fox in 1862. We know now that this eruption is of streptococcic origin. Its primary lesion is a vesicle or blister which dries into a superficial crust (Figs. 5 and 6), and there is not the œdematous condition of the skin, with pin-point vesication, nor the pruritus and sensitiveness to local irritation of eczema. Treatment by removal of the crusts and application of mild antiseptics soon clear off the eruption. Most cases of so-called eczema capitis and of eczema behind the ears are examples of impetigo contagiosa.

Another form of impetigo is that due to staphylococcic infection and known as *impetigo of Bockhart*, in which the eruption consists of pustules seated around hair-follicles. This impetigo includes the common boil, and also sycosis menti (Fig. 7) and other staphylococcic infections formerly confused with eczemas.

There are also many forms of *chronic dermatitis* of streptococcic or staphylococcic origin which are gradually being differentiated from eczema.

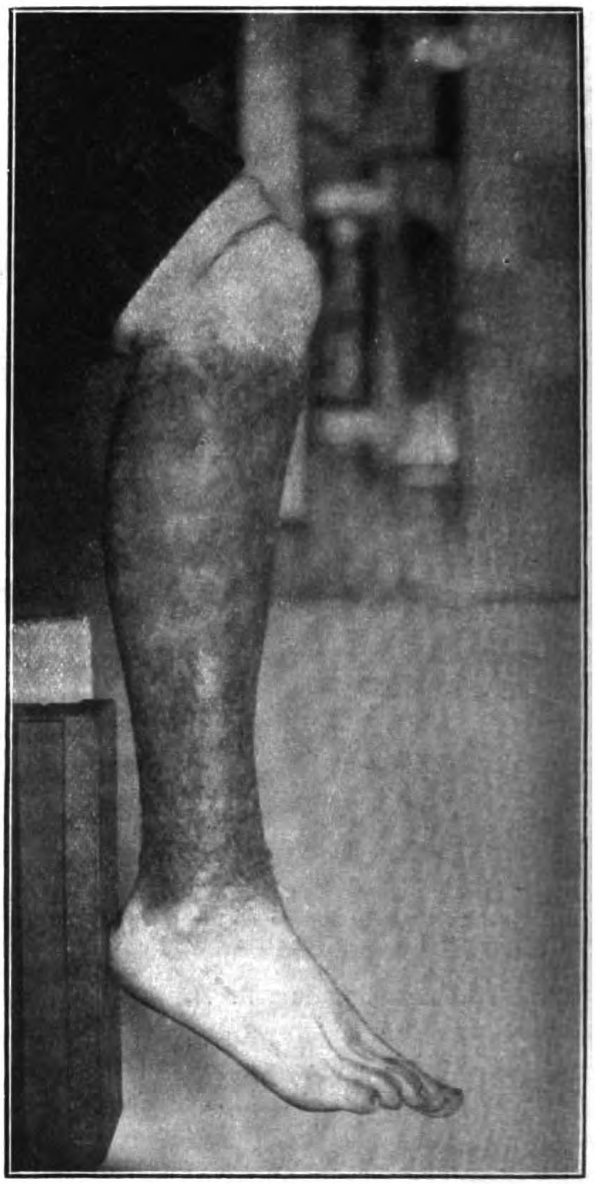

FIG. 8.—A not uncommon form of chronic coccal infection (chronic impetigo) not affected by the usual protective treatment for eczema, but cured by mild antiseptics.

Such eruptions are distinguished from eczemas by the absence of the tendency to exacerbations on irritation, by their sharp spreading margin, and by the fact that they clear up with antiseptic treatment (Fig. 8).

Another eruption which includes many types is that known as *seborrhœic eczema*. This is now

coming to be regarded as more nearly related to the impetigoes than to the eczemas. It is probably the result of infection by a mild strain of staphylococcus (*Staphylococcus epidermidis albus* [Fig. 9]).

This eruption effects particularly certain regions, as the scalp, the labio-nasal fold, the flexures, and the central parts of the chest and back. Its

FIG. 9.—One form of so-called "seborrhœic eczema." Probably a coccal infection related to the impetigo.

primary lesion is a tiny follicular papule, and large, red, scaly, sometimes oozing and crusted areas are formed by fusion of these primary lesions. The eruption clears up rapidly with mild antiseptics, especially with sulphur.

Eczematoid ringworm of the fingers and toes.—It has been lately discovered that many so-called eczemas between the fingers and toes, and especially between the toes, are ringworm infections. The simulation of ringworm is often close, but the sharp margin of the vesicating, eroded or scaly area between the toes is always suggestive of ringworm, and if the patient has, or has had, ringworm of the groin, the diagnosis is almost certain. It is to be made certain by finding the fungus in the scales.

Cheiropompholyx is a vesicular, or sometimes bullous, eruption, which affects the fingers in mild cases, and the whole hand or foot in severe cases. Both hands or both feet or all four may be affected. It is common in persons who sweat much, and is thought to be due to an irritation of a toxic sweat. It usually appears at the beginning of hot weather, and often recurs every year or every few years. It differs from eczema in that the vesicles

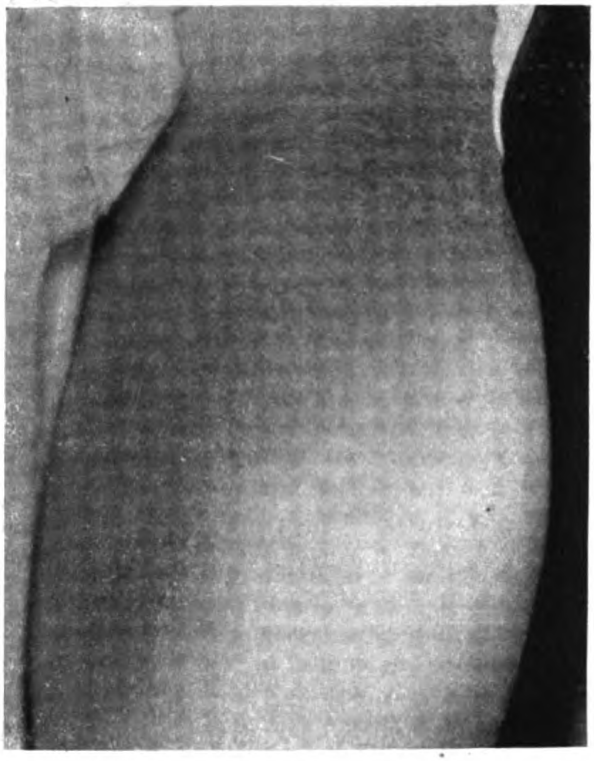

FIG. 10.—Lichenification or Lichen simplex chronicus. A long oval patch of dusky red infiltrated skin, the result of long-continued scratching. (Upper part of left thigh.)

and blisters are larger and in that there is no redness nor deep-seated œdema of the skin. Eventually the vesicles and blisters dry up spontaneously.

Lichenification.—Lichenification is the name given to areas of chronic dermatitis in which the skin is dusky red, thickened, and with the normal furrows exaggerated so as to "quadrillate" the surface of the patch. These patches are produced by long-continued scratching by the patient, and have, in consequence, always an oval shape. They often occur in neurotic persons, and start with some trifling itching spot, which the patient cannot refrain from scratching, and which he continues to scratch for years or months, until the skin becomes infiltrated (Fig. 10).

February 24th, 1913.

NOTES ON SOME FORMS OF CHRONIC ARTHRITIS MET WITH IN SEAMEN.*

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THE subject of arthritis has become a large and complicated one in the course of the last fifteen years. It was never a simple or well-understood one, and not even now are the difficulties by any means clearly and fully overcome. It has long been easy to recognise classical examples of an acute arthritis as met with in rheumatic fever and gout. In the chronic varieties of these the diagnosis is sometimes not readily made. It is safe to declare that as a result of this we may be led into error after taking great pains to be as exact as possible.

I regard it as necessary in most of these cases, and very helpful, to recognise the broad fact of a particular and inborn tendency in certain individuals towards special liability to disorders of the articular textures. This tendency runs in families as a constitutional feature. This habit of body is singularly different from another class of inborn tendency which we more readily recognise as scrofulous or strumous, in which there is great liability to infirmity of the lymphatic system, with a remarkable proclivity and vulnerability to the influence of tubercular deposition. Between these two habits of body there is a remarkable antagonism, although we have large experience of a variety of arthritis in the latter well known as strumous or tubercular arthritis—a very different condition from rheumatic or gouty arthritis.

I have discussed this matter with you in former lectures, and shall not enter further into it to-day.

Our inquiries are now pursued in a wide field, as we consider the varieties of arthritis which enter into the large category of what are commonly known as rheumatoid or osteo-arthritis. We have long known that few, if any, of these are sequels, in any correct sense, of rheumatic fever, for that malady leaves, in most cases, no malign defects in the joints, however severely involved they may have been in the acute and painful stages. It is well to recognise this much at the outset. We cannot say the same in respect of the fibro-serous and muscular structures of the heart in these cases, but we are not concerned with them now.

Following up our conception of a special liability to be involved in the component structures of the joints, we come at once in face of the modern views which compel us to regard the provoking agents of their disturbance as *toxins* derived somehow, endogenously or exogenously, which alight on these parts, and tend to pain, swellings, new growths and disability—in one word, to chronic arthritis.

The first of these toxins to be considered is that produced by infection from the inroad of gonococci. This is exogenous, of course, and arises from urethral or vaginal surfaces, afterwards permeating the system. I place this first, because we have long recognised what we used to call gonorrhœal rheumatism. Far too long, indeed, has this infection been regarded as a simple matter, readily curable and soon forgotten. Yet we know that its effects may be no less than disastrous at times, and the knowledge of this fact should be better inculcated in our young people than it commonly is.

Now, it is noteworthy that some persons of the inborn arthritic constitution or habit of body are specially apt to suffer from the severest forms of gonorrhœa. It has even been called "a touchstone for gout," so sensitive is the gouty man to gonococcal infection in a bad form.

This condition of gonorrhœal arthritis demands particular attention in this clinic, because the majority of our patients here are, or have been, sufferers from the infection which induces it. It is curious to find that these cases were formerly discussed and treated by surgeons, and few allusions to it occurred in treatises on medicine. Its specific toxic nature was gradually surmised, and now it is recognised as a constitutional disorder amongst the infections. Sailors, too, frequently suffer from imperfectly treated gonorrhœal arthritis, and consequently provide many cases of arthritis due to this source.

Its main feature is its obstinacy and persistence, and its rebelliousness to treatment. It is often mistaken for arthritis of other kinds, and unless the

* A Lecture delivered in the London School of Clinical Medicine.

source of it is sought and appropriate treatment directed to the urethral surfaces it is especially apt to linger. The earlier this recognition is made the better for the patient, and inquiry as to it is imperative in all forms of painful and disabling arthritis, especially in young adult males. In women, too, this matter demands inquiry sometimes to clear up a diagnosis. The gynæcologist can tell of grave pelvic disorders due to gonococcal infection, the source of which may be unsuspected in women.

One or many joints may be involved, the wrists, elbows and knees especially, and the sterno-clavicular and inter-vertebral joints. The plantar fascia is also apt to suffer. There is much pain, synovitis at first, fibrous adhesions later. Pus is rarely found in the joints. The synovial fringes are the chief seat of the microbes, but the fluid from a hydrarthrosis may furnish evidence of them. Early stages may resemble acute rheumatism in respect of polyarthritis, or the bursæ and synovial sheaths may be mainly affected. Chronic and painless monarthritis may be all there is to find in some cases with no local heat or redness. A hot joint is always indicative of an acute process.

Ordinary treatment for rheumatism, as by salicylates, is found to be ineffectual, and this of itself is suspicious, and should provoke inquiry at once as to an urethral focus of mischief. Iodide of potassium is hardly more effective as a therapeutic measure, and harmful if given till the primary disease is checked. We are thus without aid from any specific drug-medication in these cases. Fifty years ago Sir Thomas Watson expressed his belief that in all these chronic rheumatic disorders there was some morbid condition of the blood. To-day we know that this is indeed the case, and our modern therapeutic efforts are directed to neutralise this present matter by appropriate vaccination. This should be autogenous, and the patient's own organism is the proper remedy, prepared by culture. Small doses of from 4 to 10 million cocci are to be injected about once a week *sub cutem*, and the earlier in the case the better. In later stages where gross fibroid adhesions have been established, not much benefit is to be expected. Of course, the first step is to medicate the urethra by appropriate injections, of which potassium permanagate 1 in 4000 is useful, or protargol 10 to 25 per cent., with sandal-wood oil given internally, not omitting slight purgation with magnesium sulphate. This attacks the mischief at its source.

I have entered at some length into this variety of arthritis, because it is so largely before us in this clinic.

We do not often meet here with the pneumococcic form of arthritis. This has only been recognised as a variety since we became aware of the virulence of the specific microbe which determines it. We now know that it may prove a source of many forms of grave disease, such as pleurisy, otitis, meningitis, peritonitis, etc. The larger joints are most frequently involved, the knees, shoulders, elbows and ankles. It is a purulent arthritis, and the treatment is surgical, consisting in opening the joint and draining it. Vaccination with anti-pneumonic serum has, so far, not been found of service.

Another source of blood infection leading to arthritis is alveolar pyorrhœa. It, also, has not long been recognised. This is remarkable when we consider how many foul mouths there are in all communities. Various microbes may be found in cases of pyorrhœa of the gums—*Streptococcus salivarius*, *staphylococci*, and *Micrococcus catarrhalis*. Diffusion of any of these (and several of them may occur together) leads sometimes to rebellious acute or chronic arthritis. Vaccination from a culture of whatever is met with is an appropriate treatment, the original condition being assiduously dealt with by applications of peroxide of hydrogen, potassium chlorate, or other antiseptic mouth-washes. Not seldom many carious teeth require removal before the disorder is quelled. Here, again, an early recognition of the septic source is important.

Amongst examples of arthritis which come before us here are those due to the conditions which determine gout with their associated peccant matters. The latter are no doubt autogenous provoking toxic elements. There is no proof of any intrusive microbe in this case. In most instances there is found to be the special arthritic habit of body. Amongst seamen we have the common provocative elements of alcoholic drinks. Many of these cases are not recognised as of gouty nature, and are often regarded as examples of rheumatoid or osteo-arthritis. The diagnosis is by no means easy in many cases. Careful

observers now make a distinction between the two latter ailments. The former is regarded as more frequently occurring in the young, and the latter in elderly persons. Radiography is now employed with a view to distinguishing any one of these three arthritic conditions—gout, rheumatoid arthritis, and osteo-arthritis. I do not yet feel assured that this has been accomplished by this method. A generalised form of osteo-arthritis presents many of the characters of rheumatoid arthritis. There is less difficulty in attributing largely to mere senile changes such conditions as *morbus coxæ senilis*, spondylitis, or local arthritis, often resulting from injuries and strain. In such cases sailors and labourers are naturally more exposed to these provocations than other persons. We may be fairly certain that arthritis generally betokens some form and degree of infection alighting on predisposed textures and localities. Rheumatoid arthritis in an acute form may at first much simulate rheumatic fever. Here the joints of the extremities and the temporo maxillary are apt to be mainly involved. The cardiac structures escape. It is in the chronic form of these cases that we sometimes meet with much sweating, tachycardia, and pigmentation, especially in females, indicating a nervous element.

It is alleged that fine distinctions can be made by means of radiography between osteo-arthritic damage to joints and that met with in rheumatoid arthritis and in gout. I feel doubtful about this. Specimens of severe gouty arthritis with uratic deposits in them, when deprived of the latter by immersion in methylated spirit, which dissolves them (pure alcohol does not act in this manner), show appearances which are essentially similar to those of rheumatoid arthritis. In the latter the "lipping" and outgrowths are shown to be of cartilaginous overgrowth, while in gouty arthritis there are found to be bony changes. These lesions are probably all of a toxic nature, but the toxins differ, exogenous in the osteo-arthritic and rheumatoid, and endogenous in origin (auto-toxic) in the gouty variety. The diagnosis may often be largely aided by a full consideration of the diathetic proclivity and habits of the patient in instances which present difficulty. We have in our clinic here many cases which are not easily and distinctly referred to any of the types dogmatically set forth in the text-books. They may represent blends in respect of separate toxins For example, in one of my smaller wards there is now a young seamen suffering from chronic and painful arthritis of wrists, elbows, knees, and ankles. There is a history of gonorrhœa. The case has been tedious. The urethra has long been free from gonococci. He had symptoms of alveolar pyorrhœa. Cultures from his gums showed, according to Dr. Davies, *streptococci*, *staphylococci*, and *Micrococci catarrhalis*. One might ask which of these was the specific infecting one. A vaccine was prepared from all three, and this has been used with benefit during the past five or six weeks. It was not a promising case for vaccine treatment, but it shows how varied may be the sources of toxæmia in certain cases. Again, as Dr. Hale White has lately pointed out, we may find the source of chronic infective arthritis to be situated in the urinary tract when there may be no localising symptoms of disorder there. An examination of the urine may reveal the presence of infecting elements, and so lead to effectual therapeutic measures.

In the case of seamen we have to consider how greatly they are exposed to damp and vicissitudes of climate. They constantly wear wet clothing, and sleep in damp bunks and vitiated atmosphere. The stokers pass from high to low temperatures imperfectly clad, exhausted by muscular labour. Small wonder that these men are apt to suffer from arthritis, fibrositis, and other lingering pains, for these are just the predisposing conditions which favour the attack and progress of any specific toxic matter which may be latent in the body.

I pass now to another matter which is worthy of attention, especially in connection with our cases in this clinic, for we find, unhappily, that a large proportion of our seamen patients have been the subjects of venereal disease, commonly inadequately treated. The question arises, Has this factor any bearing on the form of arthritis we meet with here? The recent revelations which have come to light respecting the latency of syphilis by means of the Wassermann reaction at the hands of experts have to be borne in mind. This latency has long been recognised, and we have wondered where the concealment has taken place. Latent malarial impregnation has also long been known, but we have hardly yet discovered the intimate causes of it, although we recognise the modifying influences

of it on other morbid states. I doubt if we have sufficiently noted the modifying effect of latent venereal taint on other disorders, especially when there are no obvious tertiary or other lesions to direct our attention to the matter. We know little enough of any form of syphilitic arthritis. There are cases of subacute joint pains in the early secondary stage, affecting chiefly the larger joints, sometimes leading to an indolent hydrarthrosis. These are benefited by mercury and potassium iodide. In congenital cases we may find thickening of the epiphyses with osteophytes in children.

I think that we may fairly conceive some rebellious forms of arthritis in the subjects of former venereal infection to be modified as to chronicity by the dregs of this taint, and this in cases showing no obvious signs of the cachexia, just as we meet with instances of parasyphilis presenting no special signs of the original taint, or, as in the case of aneurysms where this disease is due to old venereal infection, but void often of any overt signs elsewhere of syphilis. It may be well, therefore, in some of these lingering arthritic cases, in our clinic, at all events, to apply the Wassermann test for venereal latency, and to apply appropriate medication if necessary.

There are other causes of arthritis to be mentioned. We meet with it as an associate or sequel in scarlet fever, enteric fever, variola, varicella, and dysentery, the latter only in the bacillary variety, and not in the amœbic form, and not leading to chronic changes. In all these we recognise the toxic quality pertaining to them, not omitting that due to the tubercle bacillus in the case of strumous persons when it invades the joints.

Now as to treatment. We fall back, as we too often must do, on Trousseau's maxim that "chronic diseases demand chronic therapeutics."

It is certain that the recognition of toxic elements in most cases of arthritis has led to good results in many cases by means of appropriate vaccine treatment. The earlier such a practice is begun the better. Expert bacteriology alone is of any value here. The cost of this is a serious matter in private practice, and hospital patients have greater advantages in this respect than many of the public in better positions in life.

One main object is to raise the general health of the patient to its highest level. Pain always demands rest. Locality and environment must be duly considered in all cases. A sailor's life is often one of exposure to prolonged damp and cold, to various hardships, rough fare, strain, and fatigue. These are provoking conditions for arthritis. A warm bed and blankets are needed. The diet must be appropriate to the particular case. The teeth and mouth must be carefully cleansed and kept aseptic. The urethra and urine have to be examined and rendered natural. Painful joints must be wrapped in cotton-wool and bandaged with flannel. Local applications vary in value. Belladonna liniment on cotton-wool is often soothing. One of the best applications is strong brine applied on folds of lint wrapped round the joints, covered with oiled silk, and worn all night for three weeks at least. Good results often follow this. We may employ ionisation more specifically in this way. Iodide of lithium or the salicin ion may be used, several folds of lint being applied, soaked in a solution of 2 per cent., and large electrical currents passed through these pads if possible for an hour at a time daily. Tinctures of iodine and aconite may be painted laterally on affected joints, but not encircling them.

In early stages of osteo-arthritis and *malum coxæ senile*, the actual cautery near the joint is of use. A milder form of beneficial counter-irritation is the application of Corrigan's thermic hammer at intervals. These are amongst many therapeutic agencies much forgotten in these days.

Internally, sulphur and guaiacum resin in tablets, 2½ gr. of each, may be given twice or thrice daily, or in the form of confection of sulphur with guaiacum. Cod-liver oil is often serviceable in cases manifesting cachexia and debility, and full doses of compound decoction of sarsaparilla, with guaiacum and Huxham's tincture of bark are also helpful. Stout or port wine is useful, and the latter is also of service in some cases of lingering gouty arthritis where there is debility in elderly subjects of both sexes. I will urge you to pay small heed to any *doctrinaire* systems of diet, vegetarian, anti-purin, or other in these cases, for each one is a special study in itself. In respect of climate, we find that inland fresh air is best, on a dry soil, in sheltered positions. Sea air is commonly noxious. The best results follow persistent treatment, and we must be prepared for chronicity in many of these very trying cases. The baths of Droitwich and Woodhall Spa are desirable if they can be employed. To suggest these alleviations and amenities for our patients here would be indeed a counsel of unattainable perfection. With better accommodation, better clothing, and better diet for our seamen, and with better warning as to the risks of venereal infection and the abuse of alcohol, it may be hoped and expected that the amount of arthritic disease of all kinds in sailors will steadily diminish.

February 24th, 1913.

OCCLUSION OF THE COMMON CAROTID BY A METAL BAND.

By R. ATKINSON STONEY, F.R.C.S.I.,
Visiting Surgeon to the Royal City of Dublin Hospital.

To the surgeon about to tie one of the large arteries of the body, the question of whether the collateral circulation will be sufficient for the needs of the distal tissues must always be an anxious one. Important as this question is, in the case of young individuals with healthy blood-vessels, the elasticity of whose walls is unimpaired, and with a heart whose muscular tissue is sound and able to stand any extra strain that may be thrown upon it, it is still more important in old patients with atheromatous arteries, and hearts affected by fatty degeneration or valvular disease. This danger of failure of the collateral circulation is present in the case of the large arteries of the extremities to only a slight degree, but is a very real danger in the case of the common carotid artery. Although, in recent years, since the introduction of perfect aseptic technique, and the improved method of ligaturing arteries without rupture of the inner coats, this danger is not as great as it used to be; still, even at the present day, and even in the hands of the most skilful operators, unfortunate results of ligature of this artery in elderly people are sufficiently frequent to make any method of avoiding this danger welcome. After ligature of the common carotid, symptoms may arise at once owing to sudden anæmia of the brain; these may pass off or may remain permanent. In other cases the symptoms do not arise for some hours or even days, and are then due to thombosis or embolism. If, after ligature of the common carotid, cerebral symptoms arise pointing to failure of the collateral circulation, it has always been considered that more harm than good would be done by removing the ligature, as in its application and tying a sufficient amount of injury is probably always inflicted on the intima to roughen its surface and favour the formation of clotting, and, therefore, lead either to a spreading thrombosis or embolism. To overcome this difficulty the method of occluding arteries by metal bands has been introduced. This method has been largely experimented with and very thoroughly worked out by both Halsted and Matas in America, and the latter has shown by experiment that, if a large artery is compressed by a metal band sufficiently tightly to completely occlude the lumen, three or four days will elapse before adhesive or obliterative changes in the intima occur. If, therefore, symptoms of cerebral anæmia should occur during the first three days after occlusion of the vessel, the wound might be reopened, and the metal band removed with a fair chance of complete restoration of the circulation and the disappearance of the symptoms. That the danger of ligature of the common carotid artery is still a very real one may be seen from the following passage in one of Matas' recent writings: "A long and abundant experience with the surgery of the carotid artery, amounting in twenty-five years to over seventy-eight ligatures and extirpations of the common carotid and its two branches, has fully convinced me that the risk of fatal intracranial and cerebral complications resulting from an insufficient collateral circulation is not to be under-estimated. Entirely apart from the secondary risk of sepsis the immediate dangers of cerebral ischæmia deserve the most serious consideration on the part of the surgeon."

A thin band of aluminium may be used; it should be of sufficient length to bend into the shape of an aneurysm needle, then when passed round the vessel it may be compressed till the lumen of the artery is obliterated, as shown by absence of a distal pulse, and then the excess is cut off. In the case of the common carotid artery it is most important that, if possible, the operation should be done under a local anæsthetic, so that the patient may be fully conscious at the moment of obliteration of the vessel, and at once afford prompt information of the cerebral effects, if any, of the interruption of the blood-current in the brain.

The following are brief notes of a case of extensive malignant ulceration of the tonsil and pharynx in which I occluded the common carotid artery by the application of an aluminium band under novocaine anæsthesia.

E. D— was admitted to the Royal City of Dublin Hospital on June 14th, 1911. He gave his age as 62, but he had all the appearance of being at least ten years older. There was a deep malignant ulcer involving the right tonsillar region, the lateral wall of the pharynx, and extending on to the side of the tongue; the patient said he had noticed it for some

months. It was clearly quite irremovable, but owing to the depth of the ulceration and the rapidity with which it was spreading it was considered advisable to ligature the common carotid artery with a view to both lessening the danger of a fatal hæmorrhage from erosion of the wall of a large vessel and also possibly starving and so delaying the growth. Extensive glandular involvement in the upper part of the neck would have made ligature of the external or internal carotid a very difficult, if not actually an impossible operation. The patient confessed to being a heavy drinker, his arteries were atheromatous, and his heart, though showing no symptom of valvular disease, was weak and probably fatty. Altogether he appeared a most unsuitable patient for the administration of a general anæsthetic or the performance of an operation, especially ligature of the common carotid, the weakness of the heart and the atheromatous condition of the vessels causing increased liability to danger from interference with the blood-supply to the brain. It was therefore decided to attempt to occlude the common carotid artery with a metal band under a local anæsthetic. Two thin strips of aluminium sheeting were cut about three inches long, ·4 and ·7 of a centimetre broad respectively. On June 17th he was given a hypodermic of ¼ gr. of morphia and about one hour later was brought to the operating theatre; a solution of novocaine and adrenalin was injected under the skin of the neck along the line of the carotid artery and then more deeply into the tissues of the neck. The common carotid artery was then exposed above the omohyoid muscle in the usual way, the sheath was opened and an aneurysm needle passed round the vessel; the narrower strip of aluminium was curved at the end and then passed round the artery immediately below the needle, which was then withdrawn. The metal band was then compressed slowly with the fingers till the temporal pulse was obliterated and the ends were cut short. The muscles and platysma were stitched with two layers of catgut sutures and the skin closed with Michel's clips. Throughout the operation the patient complained of no pain, and felt nothing beyond slight discomfort when strong retraction was made on the deeper tissues. There were no cerebral symptoms at the moment of occlusion of the vessel, and afterwards the patient appeared none the worse for the operation. The clips were removed on the fifth day, when the wound was found completely healed. He left hospital on June 29th, the twelfth day after operation. During his stay in hospital there was no evident increase in the size of the ulcer, and when seen about three weeks later it looked much the same; after that the patient was lost sight of, and nothing has been heard of him since.

February 24th, 1913.

CHRONIC INTESTINAL STASIS.*

(*Discussion continued from p.* 320.)

Mr. G. ROWELL said there were some details respecting anæsthesia in subjects of intestinal stasis that might be of interest. Such patients were often in an exceedingly bad general condition of health, and as the operations were often prolonged, and often involved procedures that had a shock-producing tendency, many of these operations were amongst the most formidable in surgery. Two facts commonly characterised anæsthesia in these patients. As one might fairly expect, they were particularly unable to withstand the slightest overdose of an anæsthetic, and, comparing them with persons of similar physique who had no marked symptoms of intestinal stasis, the induction of narcosis to the necessary degree was often unusually difficult. The result was that, as regarded the degree of narcosis, they had a smaller range of safety than the normal, and the depth of sleep consequently needed to be carefully regulated throughout to attain the best results. The increased difficulty of induction was, no doubt, due to their long-continued poisoning, which apparently, slowly but permanently, raised the excitability of the reflex centres—precisely as alcohol, tobacco, morphia and other poisons did, when indulged in for long periods. As these patients were never robust, this peculiarity only resulted with the method he used in a somewhat longer time than might be expected being taken up in inducing anæsthesia. Practically all of those patients that he had had to deal with for some years past had been anæsthetised by the same routine method. After a preliminary injection of morphia and atropine, ether had been given by the open method, with the transfusion of normal saline solution into one or both axillæ from, or in some cases from just before, the beginning of the administration. From three to five pints were usually given, which was accomplished in the first fifteen or twenty minutes. It was owing to Mr. Lane's wish that he first used this method for these operations, and so far as the patient's safety was concerned he was confident that no other method was to be compared with it. The morphia appeared to be chiefly of value for its mental effect

* Delivered before the Medical Society of London.

and did not shorten the period of induction. The atropine he regarded as of the greatest importance and he used it whether morphia was given or not. The addition of scopolamine appeared to make the induction easier, but it was rarely necessary. When this method was used the most useful guide to the degree of narcosis existing was the amount of movement which could be produced in the lower jaw. As one's finger was always at the angle, this information was constantly to hand. The pupils could, as a rule, be kept small throughout, except in those who were very ill indeed, when the size was a warning to keep the patient as lightly narcotised as could be permitted. The corneal reflex in these patients was always kept present, and often nystagmus also. When once the operation was well begun a much lighter sleep was permitted, which was increased at certain times as might be necessary. The procedures that demanded a slightly deeper narcosis were when the parietal peritoneum was first opened, when deep structures were pulled upon, when the rectal tube was passed, and for sewing up the wound afterwards. The patient's head should be kept slightly raised. If at any moment it was desired to quickly lessen the depth of narcosis, it was lowered a few inches, and providing the circulation was fairly good, an increased depth could be quickly obtained by raising the head a few inches while proceeding with the ether. One's aim was to keep each patient at a depth of sleep of such nicety that raising or lowering the head a few inches had an immediate effect. From this it was obvious that patients in the Trendelenburg position had to receive an unnecessary amount of ether, and the administrator was deprived of the useful help of being able to drop or to raise the head a few inches; so that, if the surgeon could do without it, it was in the patient's interest not to employ the Trendelenburg position. There were two specially dangerous types of intestinal stasis patients. The first was the young subject with an acute condition of the complaint, to whom it was by no means difficult to give too much ether when once under. The second type was the man over fifty who looked fairly well, but had atheroma. He had actually seen a man of this sort stop breathing and turn pale while quite lightly under open ether. Lowering the head and pressure on the chest and remarkably little ether afterwards enabled the operation to be performed, but after doing well for ten days or so he died somewhat suddenly, and it was found that he had a calcareous aorta. In three other similar cases he had encountered feebleness of the breathing, which constantly threatened to stop and required careful treatment. All these three cases had obvious atheroma. By the method of anæsthesia described, after-shock was rare, and, indeed, only occurred in cases that were desperately ill beforehand. If symptoms of shock threatened no drug was so useful as pituitary extract; but so rarely was this the case that, though he had constantly been anæsthetising these patients for some years, he had only used it, so far as he could ascertain, in five of them. After-sickness was slight or absent. He had emphasised the danger of anæsthesia in intestinal stasis because it was easy for an inexperienced administrator to give the most extreme cases too much anæsthetic. But by careful employment of the method he had described he had found that the worst cases could be taken through prolonged operations without any great anxiety. For many years past he had been privileged to see Mr. Lane's work on these cases, and he had observed with the keenest interest the evolution of his ideas as one fact after another came to light. He wished that time permitted him to allude in detail to the various stages by which acquired adhesions, appendicitis, large bowel obstruction, ovarian troubles, sigmoid obstruction, the ileal kink, duodenal obstruction, exophthalmic goitre, mammary degeneration, and other conditions had been brought into the clinical picture as it was presented to them today. Every month now appeared to be adding fresh facts, and he felt convinced that they were upon the threshold of such knowledge of this subject as would entitle Mr. Lane's work in this field—by its ultimate effects upon the health of mankind—to rank among the most important discoveries in modern medicine.

Neosalvarsan.—Wolff and Mulzer ('Münch. med. Woch.') have used neosalvarsan in 30 cases of syphilis. They find it far less powerful than salvarsan, and far more dangerous. They warn particularly against its use in ambulant patients. The toxic effects observed were as follows: A chill in 1 case; Herxheimer's reaction in 3 cases; headache, vomiting, and diarrhœa in 14; extensive drug eruptions in 4; herpes labialis in 2; nephritis, paralysis, and other severe symptoms in 1 case.

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ON MUCOCELE OF THE GALL-BLADDER.

By W. McADAM ECCLES, M.S., F.R.C.S.,
Surgeon to St. Bartholomew's Hospital.

Gentlemen,—I have recently had under my care two cases of mucocele of the gall-bladder, and for this reason I am bringing the facts concerning such a condition before you to-day.

The first patient was a woman, æt. 43 years, who for two years had suffered from indigestion, with a vague history some six years previously of a slight attack of jaundice. She had not noticed any tumour in her abdomen, but the examining hand quickly detected a firm, somewhat elastic, rounded swelling reaching down to the level of the umbilicus. It could not be made to go back into the loin, although it could be grasped bi-manually. It was not tender except on very deep pressure over it; it moved fairly freely on respiration, and could be moved laterally by the examining finger.

Some discussion arose as to its nature, one observer believing it to be almost certainly a tumour in connection with the right kidney, while another examiner thought it was associated with the ascending colon. Exploration showed it to be a greatly distended gall-bladder, with a single stone blocking the cystic duct. The whole bladder was removed, together with the duct for a little distance beyond the site of the impaction of the calculus. She made a very good recovery, and was quite freed of all her gastric trouble.

The second case was a much younger woman, æt. 23 years, who stated that she had been quite in her usual health until five weeks before her admission, when she had some very severe pain in the upper part of the abdomen. She did not vomit, and there was no jaundice after the attack.

A fortnight later she noticed a lump on the right side of the abdomen not far from the um-

bilicus. Again, on examination, a well-marked, firm, tense and but little tender swelling could be felt. It could be pressed more definitely back into the loin than in the former case.

Here again diagnosis was uncertain, some considering it a distended gall-bladder, others a large Riedel's lobe, and others, again, hydronephrosis. The age of the patient and the absence of hepatic symptoms and signs was against the diagnosis of a gall-bladder swelling, but laparotomy proved it to be the last. Three calculi were found in the cystic duct, which was ligatured beyond their impaction, and the gall-bladder removed as a whole. Again the patient made an uninterrupted recovery.

Mucocele of the gall-bladder is not so commonly seen as a gall-bladder distended with bile. The condition is of sufficient interest to be dealt with in some detail. In order to appreciate fully the ætiology of such a mucocele it is desirable to recall some points in the anatomy of the bile-ducts and the gall-bladder. From each lobe of the liver a duct proceeds—the right and the left hepatic duct. The junction of these forms the common hepatic duct. Not long after its formation the hepatic duct is joined by the cystic duct, this union giving rise to the common bile-duct. It is significant that a true mucocele of the gall-bladder is not, as a rule, associated with jaundice, a fact of great importance in diagnosis. A reference to the anatomy of the system readily explains the causation of the mucocele with the absence of jaundice (Fig. 1). An infection of the cystic duct and the gall-bladder occurs from the ascent of bacteria—usually colon bacilli—from the duodenum and along the common bile-duct. Bile salts, in consequence of this invasion and the activity of the organisms, are deposited from the bile within the gall-bladder, and one or more gall-stones are formed. One of these calculi, probably owing to the contraction of the muscular tissues of the bladder wall, passes into the com-

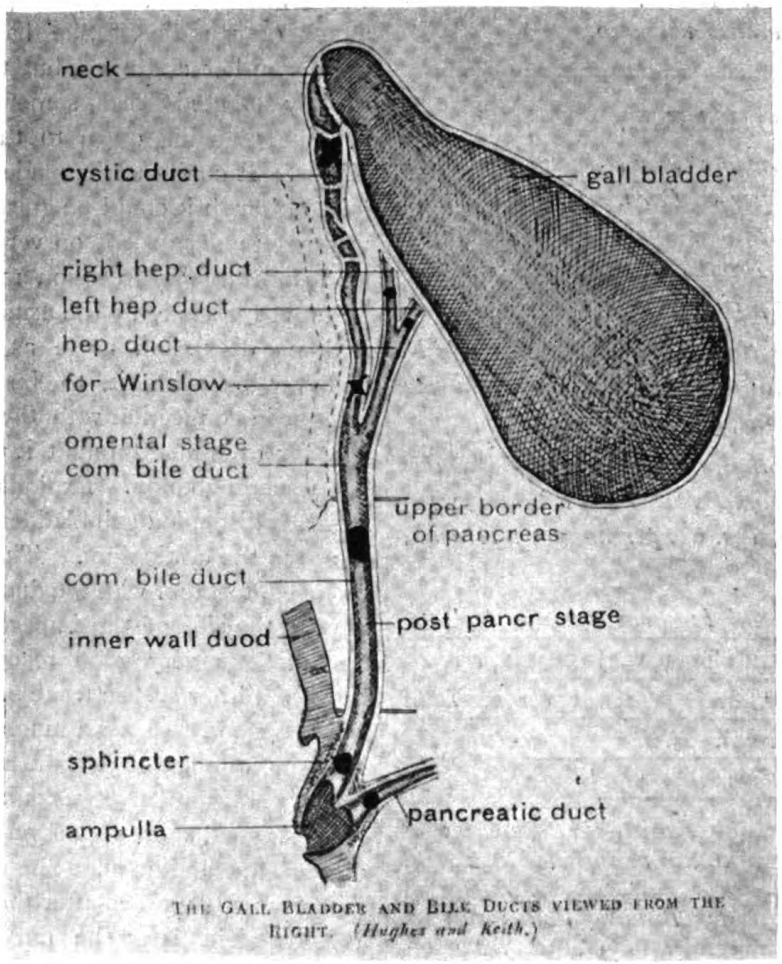

FIG. 1.—Anatomy of the biliary passages.

mencement of the cystic duct and lodges there, completely blocking its lumen. The attempt of the gall-stone to pass along the cystic duct into the common bile-duct is often attended by severe biliary colic, probably dependent upon a spasm of the muscular tissue of the bladder and duct-wall. But this colic is not accompanied or followed by jaundice, for the bile excreted by the liver can pass through the hepatic duct and the common bile-duct into the duodenum without any hindrance.

The bile which remains in the gall-bladder is quickly diluted by the secretion of pure mucus from the glands in the mucous membrane of the bladder-wall. Steadily, but very gradually, the amount of fluid is increased, and the cavity of the gall-bladder dilated. This constitutes the mucocele.

In other, but much rarer, instances a stenosis of the cystic duct is found, rather than a blockage of its interior. This contraction or obliteration is the outcome of cicatrisation after ulceration due to the previous irritation or passage of a gall-stone, or is the result of operative interference, or very rarely of congenital narrowness. Occasionally a twist or kink of the duct at the neck of the gall-bladder or an outside growth pressing upon the commencement of the cystic duct may be followed by a mucocele.

The distension of the gall-bladder as it progresses leads to the formation of a definite tumour, to be felt, first of all just below the right costal margin at the tip of the right ninth costal cartilage (Fig. 2). With further distension the tumour becomes more and more palpable, projecting beyond the lower edge of the liver. Owing to the attachments of the gall-bladder to the liver there is a tendency for the fundus to pass forwards against the posterior aspect of the anterior abdominal wall, curving much in the same way as does a growing cucumber. At the same time the antero-inferior edge of the liver may be thinned to transparency by the drag of the enlarging bladder (Fig. 3).

The tumour is smooth on the surface, its termination is rounded, and its feel is tense, or it may give a sensation of fluctuation (Fig. 4). While it moves up and down with the liver on respiration, unless fixed by adhesions to the abdominal wall, lateral and antero-posterior movement can be obtained only to quite a small extent.

Percussion over the swelling gives a dull note,

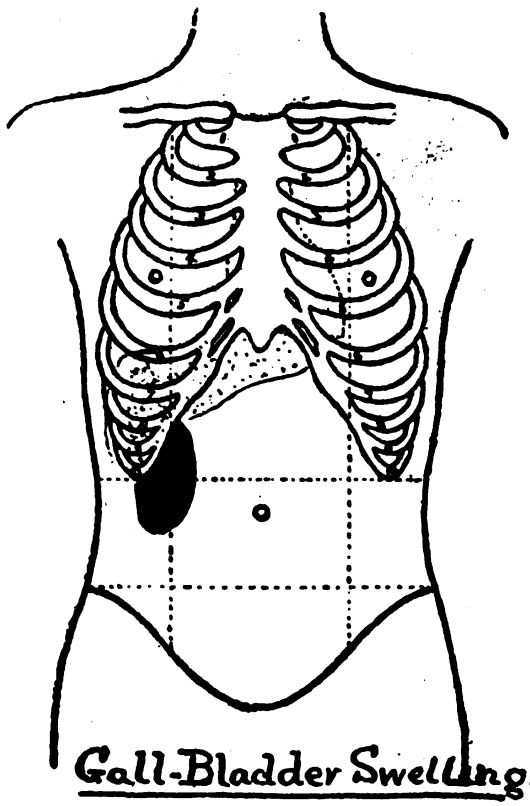

FIG. 2.—The position of a gall-bladder swelling.

the dulness being continuous with that of the liver. As a rule the swelling is painless, but if there is cholecystitis then tenderness will be experienced on pressure over the tumour, and also when the tumour meets the finger-tips pressed in below costal arch. The temperature is normal, unless a secondary inflammation occurs. Jaundice is rare, though there may be a history of a past slight and transitory attack, a history which will be useful as giving a definite indication of the probable connection of the swelling with the biliary system. A tumour presenting all the characters described above, and accompanied by jaundice, can be safely diagnosed as a gall-bladder filled with bile, due to the obstruction of the common bile-duct. If, in addition, there is evidence of cholecystitis or cholangitis the clinical picture is complete.

But several swellings occur in the region of the gall-bladder, and also are unaccompanied by jaundice, which require to be distinguished from a mucocele, and their differential diagnosis is not always easy. Such swellings are—Riedel's lobe, hydatid cyst, dilated common bile-duct, right hydronephrosis, and ovarian cystoma.

Riedel's lobe of the liver is a very interesting

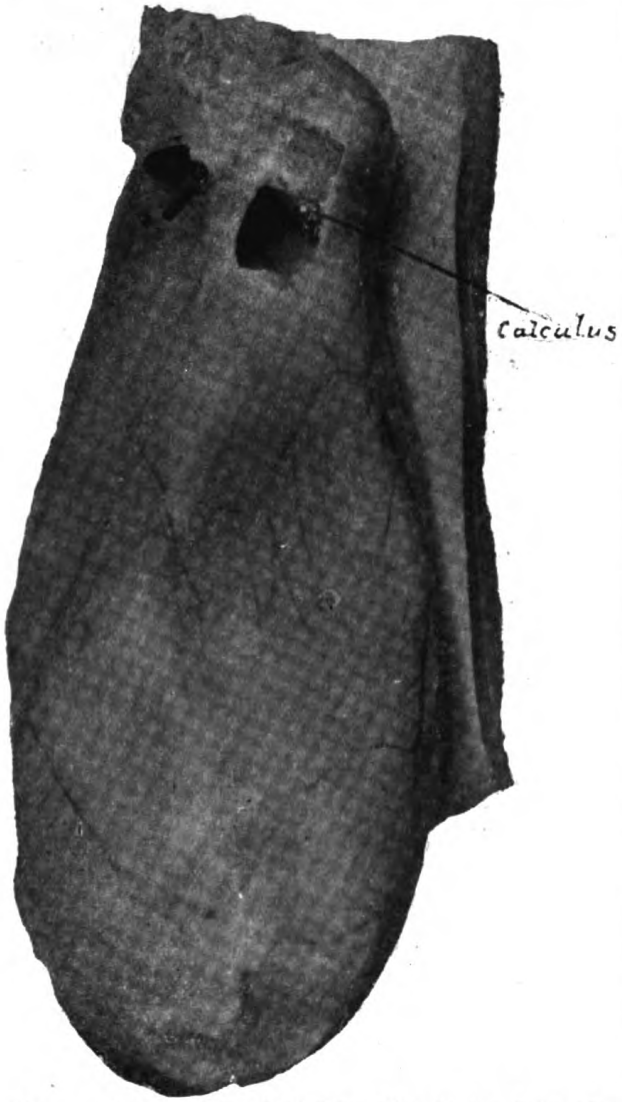

FIG. 3.—A distended gall-bladder with calculus obstructing the cystic duct.

structure. It is found much more frequently in women, and for this reason some have thought that there is a possible origin in tight-lacing, but seeing that it has been found in men, and in females in early adult life, there is probably but little, if any, connection between the two. The lobe depends from the lower edge of the right lobe of the liver as a wide, tongue-like projection situated external to the gall-bladder. Sometimes the postero-internal part of the lobe is adherent to the gall-bladder. If there is any inflammation of the latter the lobe may be mistaken for a distended, tender bladder; in fact, the diagnosis is most difficult.

The liver is a common site for a hydatid cyst, owing to the fact that the embryo of the tænia echinococcus finds its way into the portal tributaries and becomes lodged in the portal capillaries within the liver. When a cyst projects from the lower border of the liver it may simulate a distended gall-bladder, but it is usually more globular, or hemispherical, and sometimes gives a pronounced hydatid thrill. On no account should a doubtful swelling be tapped, as this proceeding may be fraught with considerable danger.

A dilated common bile-duct occasionally occurs without any dilatation of the cystic duct and gall-bladder. The swelling caused by such a lesion is more fixed, deeper, and less definite than a distended gall-bladder. A right hydronephrosis passes well back into the loin and is fixed (Fig. 5). It generally has a vertical line area of resonance over it, and is accompanied by renal symptoms.

It is quite possible for an ovarian cystoma with a long pedicle to reach so far up out of the pelvis as to come to occupy a position in which it may be mistaken for an enlarged gall-bladder (Fig. 6). The opposite confusion may also occur, and this is

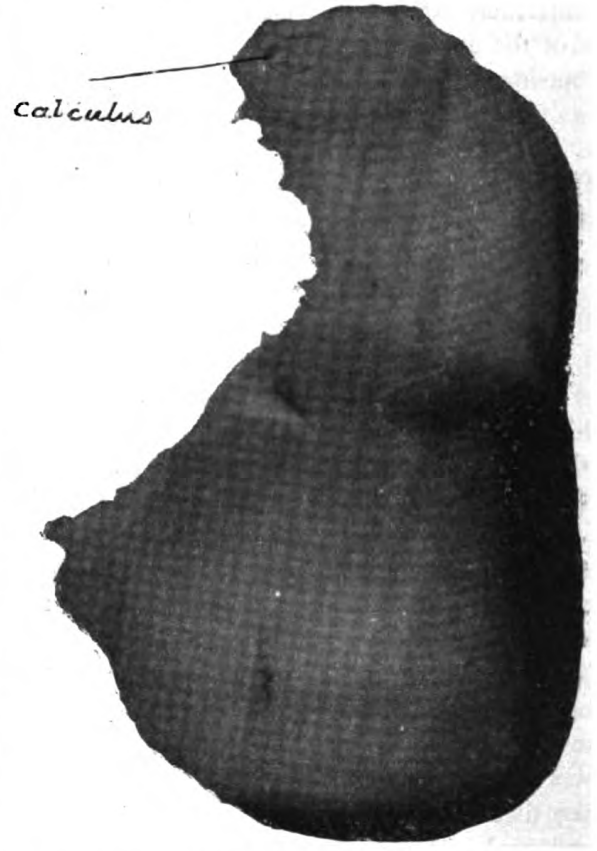

FIG. 4.—A distended gall-bladder with calculus obstructing the cystic duct.

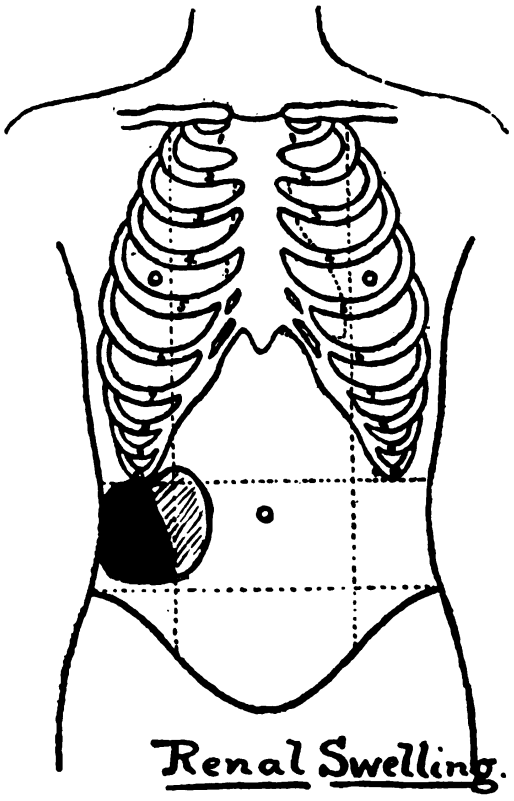

FIG. 5.—The position of a renal swelling.

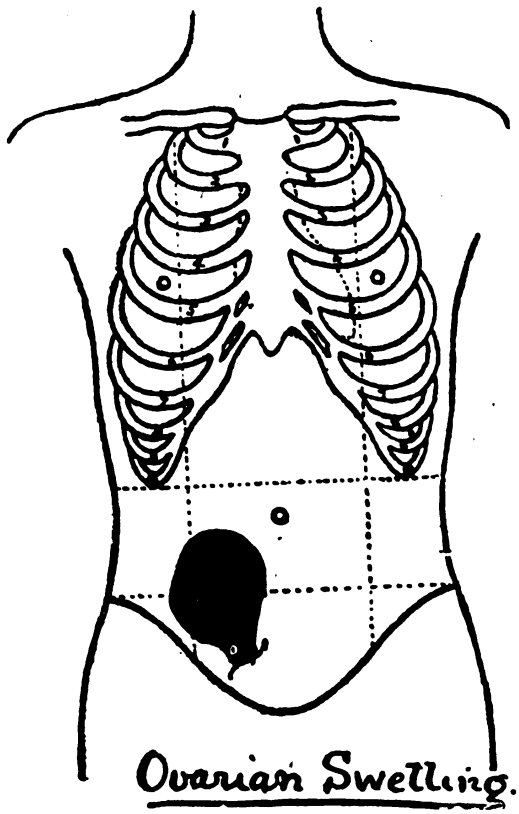

FIG. 6.—The position of an ovarian swelling.

especially so when the size of the gall-bladder is great and the liver has been pushed down by tight-lacing. The chief sign which aids diagnosis is that with a distended gall-bladder the dulness over the tumour is merged into the hepatic dulness.

In all cases of doubt in diagnosis an exploratory laparotomy is indicated.

A gall-bladder distended with mucus may rupture either spontaneously or as the result of external violence. Perforation may also occur owing to ulceration, or, in rare cases, gangrene of the wall of the bladder, or of the cystic duct at the site of the impacted stone. The extravasation of the contents of the mucocele into the peritoneal cavity may cause profound shock, and will induce peritonitis—slight if the extravasated fluid is aseptic, severe if septic.

The treatment of a mucocele when it is discovered is always by operation. The ideal method is to expose the tumour by an incision, varying in its length according to the size of the swelling, made just within the line of the right linea semilunaris. As a rule when the peritoneal cavity is reached the anterior aspect of the gall-bladder, without adhesions, is seen with a whitish appearance, small blood-vessels running over it. Further exploration soon reveals one or more gall-stones impacted in the cystic duct.

The tumour is then brought out of the abdomen, the cystic duct beyond the site of the last stone carefully determined, and there twice ligatured. It is most important to avoid any interference with the common bile-duct. The cystic duct is divided between the two ligatures. The peritoneum on either side of the lateral parts of the tumour is now separated as two flaps, and the gall-bladder lifted away from the liver substance. After its removal, generally entire, the peritoneal flaps are lightly stitched to the raw surface on the under aspect of the liver so as to avoid any subsequent adhesions to the duodenum or hepatic flexure of the colon.

The abdominal wound is closed in layers without drainage and the patient usually makes a rapid recovery, and is relieved of all troublesome symptoms. There are indications that the absence of the gall-bladder adversely affects the patient. Total extirpation of the gall-bladder with the stones in the cystic duct is always indicated in cases of mucocele.

March 3rd, 1913.

THREE FURTHER LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic; Chairman of the County of London Branch of the British Red Cross Society.

LECTURE I.

THE SOUTH AFRICAN WAR, 1899–1902.

WAR, its conquests and its failures, and the suffering involved to peaceful peasants have been so constantly in our minds of late that I have yielded to the invitation of some of you to continue my lectures on the treatment of the sick and wounded in war time, and I also hope to show you what is being done in recent years to preserve the health of the soldiers and sailors, on whose physical efficiency so much depends.

In my last lectures we noted that the care of the sick and wounded was a comparatively modern development in warfare, and that so lately as in the Napoleonic campaigns no proper provision was made for dealing with this question, which was greatly left to the chance charity of local inhabitants. The letters of the first great war correspondent, William Howard Russell, from the Crimea, attracted people's attention to the inadequate measures then adopted to lessen the sufferings of the sick and wounded soldiers, and from that time to this a steady improvement has taken place among the more civilised nations, and when official means have been exhausted, voluntary aid has stepped in and done its best to pay the debt of gratitude to those who are ready to lay down their lives for their country. But in our sympathy with the sufferers it must never be forgotten that the ultimate object of an army in the field is the overthrow of the enemy, and no considerations of pity for the necessary suffering involved must for one moment stand in the way of the accomplishment of that object.

Those who are watching events from a far distance are a little apt to lose sight of this very elementary fact, and England is seldom behindhand in a somewhat excessive pity for the sufferings of others.

The lesson which past campaigns have taught us is that, just as the general in command of the forces has to bring all his best efforts to bear in circumventing and resisting the enemy in the field, so the medical officers of the army have another and a far more deadly foe to evade, and, when he cannot be evaded, to conquer. For microbic disease is a much more insidious enemy than any we have to face in the open, a deadly foe which lurks in the stream to which the parched soldier rushes headlong at the end of a long and dusty march, which lies in wait in the deserted camp where others have bivouacked, which is blown into the mouths and throats of the men in the dust storm, and may sleep in the blanket with which they cover themselves when tired out at night. Until lately we have despised this enemy, or we have submitted to his ravages as a necessary accompaniment of war, for we have not sought to fight him as we should.

A fact not easily realised by those who have not come into actual contact with war is the enormous preponderance of cases of sickness compared with wounds which has occurred in every campaign since statistics have been compiled, especially in long protracted wars, with the one notable exception of the Russo-Japanese war, of which I am going to speak to-morrow.

Sir Thomas Longmore tells us that the average in the wars of the last 200 years gives a mortality of four deaths from disease to one death from wounds. Many of you will recall Russell's account of how new drafts of men kept pouring into the Crimea, full of martial ardour, and died from exposure, neglect and infectious diseases before they had fired a shot for their country. In the Russo-Turkish war of 1877–8, 20,000 died of bullets and 80,000 from sickness. The American Civil war had a proportion of about three deaths from disease to one from wounds. In the French expedition to Madagascar in 1895 the astonishing figures are quoted of 29 killed in action and 7000 dead from disease, while the United States Army in the Spanish-American war has the disgraceful record of 14 dead from disease to every one killed in action, though the war lasted little more than six weeks.

When the ultimatum from the Government of the Transvaal was received in England on October 10th, 1899, and war was declared on the following day, the medical arrangements for a short campaign

* Gresham Lectures.

had been thought out and had already been partly carried out. The stock of medical stores in Natal had been quietly and steadily increased during the months following the Bloemfontein Conference, and a scheme for the mobilisation of the personnel in Natal had been put forward. Additional stores and hospital equipment had been provided, and reinforcements of officers and men of the Army Medical Service were arriving from England. But as it turned out, the length of the campaign, the immense difficulties of transport and the wide distribution of troops soon proved that these preparations were entirely inadequate for the strain placed upon the army.

The probable sick-rate of any campaign depends, of course, largely upon the local conditions at the seat of war, and each military expedition has its special dangers from the medical point of view. Before the outbreak of war, attention had been called by medical men throughout South Africa to the prevalence of enteric fever, so common among all Europeans, but this special danger had apparently not been sufficiently foreseen by the medical authorities, and the loss of good fighting material and the misery and expense of a high sick-rate were the inevitable results.

But although more preventive work might have been done, no one can minimise the difficulties which faced the authorities. They had to endeavour to keep an army healthy in a country where no provision whatever was made for cleanliness, where the water supply was scanty and bad, where sudden and extreme changes of temperature were common, and where typhoid fever was endemic. Add to this the constant difficulty of persuading the men and even the officers to take ordinary precautions against illness, or to believe in the dangers to health lurking everywhere.

In his report on the medical arrangements in the South African war, Surgeon-General Sir William Wilson, late Principal Medical Officer of the campaign, speaking of this attitude of mind, says: "There is no doubt that there was a certain feeling that it is unmanly to be afraid of drinking any water, that the possibility of contracting disease was one of the chances of war, and could not be shirked. This was apparent among officers as well as men, and the obvious remedy is a little instruction of elementary hygiene, with a special application as regards duty towards one's neighbour."

It is, perhaps, hard to define the line between over-anxiety about one's bodily well-being and the common-sense view which everyone ought to have that illness is a blot in life, rendering the victim a temporary nuisance and a useless member of society. When, as in war, every additional healthy man means just so much additional fighting strength, and every sick man so much additional useless and dangerous burden, it ought not to be difficult to impress upon the minds of every reasonable being the fact that he is committing a crime who endangers his own health and that of his comrades by an act of carelessness.

A great deal is now being done to educate the officers, non-commissioned officers and men in elementary military hygiene, and when the time comes for us to engage once more in a great war we must hope that this teaching will have produced a wiser attitude of mind among all those engaged. The question has arisen as to whether a more cautious attitude in the soldier may not affect his fighting power—whether the devil-may-care fellow who scorns all precautions is not more fearless in fighting the enemy. Possibly, when a certain type of man realises the immense dangers to health in a campaign, he may become afraid to face a war at all. If so, he is not wanted. The case is somewhat analogous, it has been pointed out, to the instruction the men now get in making an attack in extended order when they are told to take advantage of all available cover. Here and there certain individuals have displayed too great an affection for cover, but on the whole this has not been the result. "The quiet, manly soldier of the present day," writes Lieut.-Colonel Simpson of the R.A.M.C., "has done as good work as the drunken, dissolute men of Flanders and the Peninsula. When the unremitting individual war against disease becomes subconscious, the effect on the mental attitude of the soldier disappears. Our aim should be to develop the intelligence of the soldier—by no means of a low grade—and, without dry-nursing him, to induce a habit of sanitary cleanliness which is as automatic as his response to the word of command on the parade ground."

Let us consider the actual conditions met with in the field and the special conditions of the campaign in South Africa. Here was an army, consisting mostly of young men, unprotected by previous experience of campaigning or of the climatic con-

ditions of Africa, pouring into a country consisting chiefly of an arid, treeless plain, broken by kopjes and dongas, very dusty and very hot. The nights were often bitterly cold, and there were frequent thunderstorms, accompanied by torrents of rain, from which the men had little or no means of protecting themselves.

Though the medical arrangements might have sufficed for the small and short campaign which was anticipated, the department was hampered by an insufficiency of personnel, an inadequate hospital accommodation, and the almost insuperable difficulties of transport which became evident when it was realised what a slim and strong enemy we had to face, and how great and protracted the operations against him would have to be.

The medical officers in charge of the health of an army are now not only expected to deal with wounds and sickness when they occur, they must be the watchdogs to guard against the entry of disease; their work must be preventive if it is to be efficient. Of all diseases, the one most feared by European medical officers in a campaign is typhoid fever, and, next to typhoid, dysentery. The South African war amply proves that these two diseases were the most important features of its medical history, for 45 per cent. of the deaths from all causes (including killed in action), or 86 per cent. of the deaths from disease, were due, according to the official reports, to "continued fevers and dysentery." The predominance of epidemic disease is not peculiar to our army; it affects every army in the field, and we may congratulate ourselves at least on the fact that we lost a smaller percentage of men from typhoid fever than has ever been the case in any previous war. Although this was so much to the good, yet 8022 men died from enteric fever alone, 19,454 were invalided, and 30,208 were either returned to duty or they were otherwise discharged. It is unnecessary to dwell upon the waste of strength and money of these 57,684 cases of enteric fever. The average number of days under treatment of a case of enteric fever was fifty-six, and when it is considered what careful nursing each case required, what difficulties the disinfection entailed, and how grave a source of danger each sick man was to those about him, it is obvious that no effort must be spared to avoid this enormous number of cases in the future.

Enteric fever is conveyed from man to man by a bacillus which is found to survive in water for many days, which can live in dust or on any bedding or clothing soiled by an enteric patient.

Flies, those inevitable harbingers of filth, have been proved to be the carriers of infection, conveying the bacilli on their feet and wings or in their intestinal tract, from which they deposit the living bacilli on food which is to be eaten by man. These pests followed the army from place to place, settled on food wherever it was to be found, and haunted the canteens of the troops. It has been surmised that it was the vast numbers present in the cavalry brigades which caused those troops to suffer more than others from enteric fever. "Where large numbers of flies were destroyed in tents and subsequently trodden under foot," writes Colonel Simpson, "a horrible fœtid odour of putrescent matter was noticed. No conception of the vast number of flies can be formed by ordinary experience; it was common to see the insides of tents black with them, and so was the food, if not protected by sacking, as was recommended by the medical authorities." The presence of these pests alone could have accounted for the spread of typhoid infection, for this disease is never absent from South Africa, and attacks both natives and Boers in large numbers every year.

The natives were known to have spread the infection along the main lines of traffic and on the Kaffir paths when they returned to their homes from the mines. In a country where the water is as scarce as in South Africa the paths and roads must necessarily lie chiefly along the river banks, where the armies were also forced to march because they found there their only water supply. A large number of natives also moved with the armies as carriers and porters, and however successful the medical authorities might be in cautioning the soldiers against the dangers of unhygienic habits, to the native mind, free from the most elementary ideas of decency and cleanliness, such teaching was quite incomprehensible.

But it was not the coloured men only who had to be feared: enteric fever broke out in the Boer laagers, and when our troops entered these camps they found a condition of indescribable filth, which doubtless was an important factor in the spread of the disease. Fatigue, exposure and privation are predisposing influences which make the soldier

especially susceptible to any infection, and these are almost inevitable conditions which have to be faced on a campaign.

But the greatest source of danger which the soldier in South Africa encountered was undoubtedly the water supply. It was known to every medical officer and to most laymen that there lay the most probable cause of infection, but the conditions were such that all precautions were rendered useless and a clean water supply became an insoluble problem. The rivers in South Africa vary enormously in volume between the dry and the wet season, becoming rapid torrents after the rains and shrinking to mere trickling streams in the hot weather. When this occurs the banks are laid bare and become the receptacles of every kind of filth and rubbish, which is swept into the stream as the water rises, so that it becomes polluted to an ever-increasing degree as it passes from one set of human habitations to another. "In standing camp," the report of the Committee of Field Sanitation tells us, "during the latter part of the campaign, all water was boiled; in hospitals and messes Berkefeld filters were also used. If boiling is properly carried out it requires large quantities of fuel, which is a great difficulty in a treeless country like South Africa. The habit of drinking water on a march is a bad one, and might with practice be controlled. It is often due to the use of strong tobacco. It is found practically impossible to prevent men drinking foul water on the march. Great care was taken to select the best possible source; medical officers went on ahead of the column to select the place for filling the water-carts. Sentries were posted to prevent animals and men, especially natives, from fouling the supply, and a European was placed in charge of the carts to see that they were filled at the selected spots. All this was well carried out in camp, and also when the columns arrived on the camping ground sufficiently early, but frequently it was after dark, and possibly pouring with rain, when the carts were probably filled at the nearest place. Then, as fire and lights were not allowed if the enemy were in the vicinity, it was impossible to boil water, cook food or dig latrines. Towards the end of the summer water became scarce, and the choice of camping grounds was more restricted, consequently the same ground was repeatedly occupied and the surroundings became more contaminated. Diseased and exhausted animals invariably make for water, and not having strength left to extricate themselves from the mud, lie down and die, so where the banks of the stream are steep the mortality is very great. The Modder River, in particular, was full of the carcases of animals."

There was more enteric fever among the English troops, both officers and men, than among the Colonials, which may be partly due to the fact that these had a larger proportion of seasoned men, men who had lived a harder life and were less susceptible to exposure and fatigue, partly, perhaps, because Colonials as a whole drink more tea than water. In the latter part of the campaign this became very general among the troops whenever it was possible to provide fuel to boil water.

All through the war it was the British regulars and volunteers who were the only troops to make systematic attempts at satisfactory sanitary arrangements. "The Colonial troops," says Colonel Simpson, "were careless in this respect, and the irregular corps were absolutely impossible."

Although enteric fever caused the greatest harm among the troops, dysentery did not fall far short of it, for there were 38,108 cases, with 1342 deaths. The great variations in temperature, causing chills, the extremely tough meat which the soldiers received as rations, besides the very bad condition of the teeth of a large proportion of the men towards the end of the campaign, tended to produce chronic irritability of the intestines, which made them very susceptible to this infection. As the number of cases increased the sources of infection became very numerous, for the mode of propagation is, as far as we know, similar to that of enteric fever.

Great as were the dangers of infection to field forces, the risks run by the besieged garrison in Ladysmith were infinitely greater, and caused the keenest anxiety to the medical officers—an anxiety fully justified by the course of events.

As a rule the field forces were well fed. "The troops always received full rations, and frequently fresh bread and meat," says Sir Thomas Gallwey in his report on the Natal field force. "After a period of specially arduous service an endeavour was always made while the troops were resting to issue fresh bread and meat and vegetables, such as onions. The benefit to the men who had been living for days on bully beef and biscuit was note-

worthy." But in Ladysmith the conditions were very different. The siege began on November 2nd, 1899, and there was a reserve store of food, bread stuff, meat, and groceries, to last from 46 to 65 days for a force of about 15,600 Europeans and natives, with some additional food stuffs in the town, which were requisitioned for general distribution. As a matter of fact the civil population had also to be provided for, and they brought the total numbers up to 21,000 men, and instead of lasting from 40 to 60 days the siege lasted 120 days, till March 1st, when it was raised by Sir Redvers Buller.

As the date of relief became more problematical rations were greatly reduced, horse-flesh was added, and an exact calculation was made to enable the garrison to exist till March 31st, till, as Sir Edward Ward reported, the men became very reduced in strength. "They had none of the things that were really necessary; they had no vegetables or things of that sort, and no butchers' meat practically. They were really living on maize flour and various expedients made out of horses." These expedients were chevril, a strong soup made from horse-flesh, which was issued nightly to the troops, a condensed form of chevril, which was a strong meat extract, chevril paste, of which each man received one ounce as a ration, and which was much appreciated, as well as a jelly similar to calf's-foot jelly, and the boiled meat which was issued as an extra. Besides a deficiency in quantity there was a lack of nitrogenous food, and an entire absence of fruit and fresh vegetables, which proved very detrimental to the health of the garrison. The rations from the middle of January to the end of the siege consisted only of three small biscuits and one and a quarter pounds of horse or mule flesh or its equivalent. Under these circumstances it was not surprising that some scurvy appeared among the men, while the depressing influences of the siege and the damp and cold were strong predisposing causes. "We noticed," writes Sir Edward Ward, "as General Buller's forces approached us the sick rate went down; as they retired it went up again." There is no doubt that men will try and keep out of hospital as long as there is something going on, something to look forward to. It is when failure and hope deferred are predominant that they feel the weight of their bodily troubles. But above all, the bad water supply of the beleagured city constituted its greatest internal danger. The water was drawn from the Klip river, which resembled other South African rivers in its changing seasonal volume. In the early months of 1899, before the war, enteric fever had been prevalent in Natal, and a considerable outbreak had occurred in Ladysmith itself, and in the hot season of 1897, 1898 and 1899 the area drained by the Klip river had been infected, and there was considerable sewage contamination of the stream. After these outbreaks some efforts were made to improve the sanitary conditions of the town, and a new intake of the water supply was made higher up the river, but this supply proved insufficient when the population crowded into the narrow area, and matters were further complicated by the enemy promptly cutting off this improved supply at the beginning of the investment. Filters, tanks and pumps, ordered hurriedly from England, arrived too late. Thus, when Ladysmith was beleaguered, it was a city ripe for an epidemic, with three dangerous factors present: (1) a bad water supply, (2) a perfectly inadequate method of dealing with sewage, (3) soil previously polluted and infected by enteric fever. As long as the siege lasted the water had perforce to be taken from the old intake, which lay in a part of the river polluted by the drainage from the town and from the Boer lines. Valiant efforts were made to mend matters. Engineers from H.M.S. "Powerful" and some railway men constructed a condensing plant with some locomotive boilers, but as fuel became scarce the supply had to be limited; rough filters were made, and as long as fuel lasted the water was boiled, and officers noted an apparent improvement in the health of the men so long as such measures could be used, but they could only be very partially adopted. It was only in the hospital at Intombe that a continuous system of filtering the water through Berkefeld filters could be kept up. During the 120 days of the siege there were 1280 cases of enteric fever with 360 deaths, and 1841 cases of dysentery with 105 deaths. The high death-rate was greatly due to the scarcity of milk and other suitable nourishment.

The arrangements made for hospital accommodation before the siege began proved to be impracticable, for the enemy mounted some long-range guns which could shell the town and were bound to inflict much loss of life in the hospitals

themselves. Representations were therefore made to the Commandant-General of the Boer army that he was shelling the wounded, and it was arranged that a piece of ground at Intombe, about four or five miles from Ladysmith, should be set apart for a hospital and for a camp for civilians, that all the sick and wounded men in hospital should be transferred there, and that a train should run daily to transport invalids and to supply the camp. Twenty-four hours were allowed by the Boers for this transfer, during which time a large camp hospital had to be established, equipped with supplies of all sorts and filled with sick and wounded. To these the Boers added ninety-four wounded men who had been left at Dundee and the personnel of the hospitals from the Glencoe columns. The medical equipment belonging to them had been taken from them by the Boers. As time went on and the number of serious cases increased, the greatest difficulty was experienced in providing doctors, nurses and orderlies, besides hospital stores. Civilians were enlisted and convalescent soldiers helped each other, but of the forty-eight medical officers five died, ten had severe attacks of enteric fever and twelve others were incapacitated, which shows what a strain was thrown upon those who could remain at their posts. On the relief of Ladysmith there were 2000 sick and wounded in the hospitals, and at least as many more who were only fit to be invalided.

Sir Frederick Treves was among the first to enter the city with the relieving force, and this is what he says: "The men were piteous to see. They were thin and hollow-eyed, and had about them an air of utter lassitude and weariness. Some were greatly emaciated, nearly all were pale, nearly all were silent. They had exhausted every topic of conversation, it would seem, and were too feeble to discuss even their relief. Ladysmith was reached at 2.30 p.m., and the food convoy did not arrive until late the same evening, so we had the sad opportunity of seeing Ladysmith still unrelieved—unrelieved so far as the misery of hunger was concerned. I had no food at my disposal, but I had fortunately a good quantity of tobacco, which was doled out in pipefuls so long as the supply lasted. It would have taken many pounds, however, to satisfy the eager, wasted, trembling hands which were thrust forward on a chance of getting a fragment of the weed. Streets were empty of all but a few tired and listless men, stores without goods, shops without customers, a railway station without passengers, a post office without letters, stamps or post-cards. No words, indeed, can fully describe this city of desolation, this little colony of the almost hopeless, this poor battered, worn out, hungry town of Ladysmith, with a bright summer sun making mockery of its dismal streets. The wretchedness of the place was not mitigated by the horrible smells which greeted one at every corner, nor by the miserable, dirty river which crawled slimily through the place."

The relieving force, which had endured great exposure, and had spent a fortnight in the horribly insanitary positions lately occupied by the Boers, at once added an enormous number of sick to the garrison.

Whilst enteric fever and dysentery were raging in Ladysmith, and among the troops marching to the relief of that town, the same dread disease was soon to fill the hospitals, and tax to the utmost the strength of the medical units with the troops which entered Bloemfontein. The weather in February, 1900, was hot, and supplies were short, there were constant encounters with the enemy as the troops were marching from the south, resulting in some casualties, and when the British forces entered Jacobsdal, on the 15th, they found two churches in the town filled with Boers suffering from enteric fever. This showed that infection was present, but the difficulty of getting any water at all made precautions in that direction almost impossible. The supply was so scanty that the line of advance was greatly determined by the course of the Riet and the Modder rivers, although the water was known to be contaminated by the Boer laagers.

As the forced marches continued tents had to be pitched for the relief of about 130 men who collapsed from the effect of the heat. On February 16th one brigade was marching and fighting for more than fifteen hours, the men carrying what supplies they could with them. The day before 170 waggons containing rations for 150,000 men and 500 cattle for slaughter had been lost at Watervaal drift, and this greatly reduced the already short supplies of food. Indescribable fatigue, insufficiency of food and bad water all tended to sap the strength of the troops and rendered them liable to infection, and the medical units had the greatest difficulty in keeping up with

the rest of the army, owing to the exhausted condition of the transport animals.

Though Lord Roberts had succeeded in driving Cronje from his lines and had relieved Kimberley the wily old Boer was not yet beaten, and had ensconced himself in his laager at Paardeberg. By dint of strenuous exertion and forced marches the British brought up sufficient forces to surround him, and to make an attack on February 19th. The battle was begun when the men had been marching at full speed for twenty-four hours; some had had no sleep, none more than a few hours, and whilst some had been able to snatch a hurried meal, many had eaten nothing. They fought for twelve hours and had 1262 casualties before they were obliged to retreat. Tents, food and blankets and every kind of medical comfort ran short, for the transport difficulties were immense along a single line of railway with bad engines and with the line constantly damaged by the enemy. The military needs, ammunition and food had to be thought of first; the medical supplies were necessarily only a secondary consideration. Cronje surrendered a week later, and when the British troops took possession of the Boer laager they found 160 sick and wounded, including ten cases of enteric fever. The prisoners stated that enteric fever had been prevalent and had killed many among them, and the laager was indescribably filthy. The water intake of the army lay down stream from this foul spot, and there was no fuel available for boiling water, while filters were useless, because the water was too thick to pass through them. Such, then, was the condition of things on the march towards Bloemfontein, which was reached in the beginning of March, after which 30,000 men were camped in and near the town.

The capital of the Orange Free State lies in an open plain, with the Modder River some three miles to the north and the hills of Thabanchu twenty-one miles to the east, where the Free Staters had been driven unwillingly to erect some water-works, because for years past the town suffered annually from outbreaks of fever. Bloemfontein takes its name from a spring in the centre of the town, which, though once considered perfect, had of late years become greatly contaminated by the filthy habits of the people, by neighbouring cesspools, and by the complete absence of all drainage.

One of Lord Roberts's first cares was to secure the water-works, and for this he sent there an unfortunately quite inadequate force of 300 mounted infantry. On March 31st de Wet with a greatly superior force made a night march, surprised the mounted infantry and a detachment and convoy under Colonel Broadwood and took possession of the hills and the works. This act, disastrous as it was from every point of view, proved a veritable catastrophe, affecting as it did the health of hundreds of valuable lives, and leading to a heavy death-roll. It was not till April 23rd that General Ian Hamilton succeeded in re-capturing the water-works, which were then found to be out of order, so that the supply from them could not be resumed till May 10th. Meanwhile the troops had mostly to depend on the shallow surface wells, known to be polluted, while a limited number only could be supplied with a better quality of water drawn from a pure spring and from a bore hole. Within ten days of the entry into Bloemfontein there were 1000 men in hospital, a large percentage of whom were cases of enteric fever, which had been contracted on the march and at Paardeberg. During the twenty weeks from March 16th to July 27th there were 1134 deaths in Bloemfontein, of which 964, or 85 per cent., were due to enteric fever. Military necessities, excessive fatigue and privation had played their part in preparing the soil for the entry of infection, but the lack of a pure water supply was the primary cause of the excessive sickness.

The military authorities took the lesson of these irretrievable disasters to heart, and great efforts are to-day being made to give adequate instruction to officers and men. Regimental and camp sanitation must be made the responsibility of every individual of the force. However excellent the hygienic laws made by the medical officers, the execution of them lies in the hands of the soldier, "whose instincts are to avoid all labour which is not evidently and immediately useful." If he is taught the necessity of sanitary cleanliness there is no reason to doubt that he will conform to the rules laid down in his interest.

"We have succeeded," says Colonel Simpson, "in the analogous case of personal cleanliness. Custom, discipline and increasing facilities have made the soldier a clean man—how clean can best be determined by those who have had the

opportunity of seeing the reserve man or old soldier among his civilian fellow workmen. The habit of cleanliness was so fixed, at least in many corps, that it withstood the trials of war, and the soldier was as clean as his officer. We must attain the same standard in sanitary matters. As a general rule, the smarter the corps the better its interior economy, and the higher its standard of personal and sanitary cleanliness. Within the range of one's personal observation, in only one smart regiment was the interior economy, the care of the men, below the standard; and that regiment's reputation suffered severely in South Africa."

As a precautionary measure against typhoid fever, it is desirable that every man should be adequately inoculated against this disease. Anti-typhoid vaccination was not so well understood thirteen years ago as it is now, and there is little doubt that it was ineffectively carried out in many instances on the soldiers going to South Africa. Also, the soldiers were not very willing to submit to the discomfort of it, having no great faith in it. We now know that anti-typhoid vaccination should be performed on two separate occasions at intervals of about ten days, but this was not generally known till after the war. Yet, in spite of this, the general analysis of the campaign shows that typhoid fever was twice as common in the non-inoculated as in the inoculated.

As the war became more extended and casualties and sickness increased, it became obvious that the Army Medical Department was unable to cope unassisted with the enormous numbers to be dealt with, and voluntary aid was encouraged. The Central British Red Cross Committee became very busy in organising and despatching hospitals, personnel and stores. Among many other organisations the Imperial Yeomanry Hospitals Committee was formed with the intention of providing special hospital accommodation for the Yeomanry in the field. As far as was practicable this intention was carried out, but it was not possible to reserve them solely for that branch of the army. The hospitals were lavishly equipped from the charitable fund, and they did splendid work for the sick and wounded. £145,000 was collected in England by a committee of ladies presided over by the Countess Howe.

A large base hospital was first established in March, 1900, at Deelfontein, in Cape Colony, for 1000 beds, and later, in the summer of the same year, the Yeomanry Hospitals Committee was petitioned to send out another one for the accommodation of at least 400 men in the Transvaal, and it was then that Dr. Langdon Brown and I had the good fortune to be chosen to go out and open this new hospital. I started at twenty-four hours' notice from Southampton with three other doctors, twelve hospital sisters and eighty-one civilian orderlies. We occupied the voyage to Cape Town in carrying out inoculations against typhoid fever, in preparing lists for the division of our future work, and in practising the orderlies in army and stretcher drill and in giving them lectures on the treatment of minor medicine, surgery and nursing. I also classified the orderlies with regard to the work for which their past occupations best fitted them. We arrived at Cape Town on July 17th, and a week later we were reinforced by the arrival of six doctors, twenty-eight sisters and eight ward maids. There was considerable difficulty in getting accommodation in the trains going north, for the single line was packed with food-stuffs, clothing and soldiers, and the only chance of travelling was often to lie in an open truck on the top of the supply stores. After leaving Kroonstad we entered the zone of de Wet's operations, and the rails, taken up and twisted every two or three miles, the culverts and bridges blown up, and the telegraph wires broken or mended were evidence of his activity. The veld was burnt in many places to show up the khaki of our men. Our train was carrying £120,000 in cash, and the last time an attempt was made to send this money the Boers got wind of it and wrecked the train. This time they only succeeded in removing some of the rails about an hour *after* we had passed Rhenoster.

Upon reaching Pretoria I was at once sent by the military Governor to commandeer a house and estate of about 150 acres, which had been chosen by Lord Roberts as the site of the Imperial Yeomanry General Hospital. I had no compunction in dislodging the owner, a colonial with a crafty face, for he had been plotting with the Boers while pretending to be friends with the English. I told him to remove his billiard table and most of his furniture, and to leave the pictures, kitchen utensils and other things which might be useful to us. Lord Roberts had already told the Royal Engineers to lay down a water supply for us, and the electrical

engineers agreed to put up an electric lighting plant. This seemed the only solution for the lighting difficulty, for there was no possibility of buying paraffin, candles or matches. When we struck a match in Pretoria to light a cigarette we solemnly waited to do so till others were ready to participate, for matches were exceedingly precious. We next planned out the future camp, and before laying it out we had to procure Kaffir boys to clear the ground by burning the long grass. This they did very cleverly, previously arming themselves with sacks dipped in water with which they controlled the flames. For some days, until the arrival of Surgeon-Major Kilkelly, I had to act as military commandant, and as our stores consisted of little besides marquees, tents and bedding I had to procure much of the hospital equipment in Pretoria and Johannesburg. We arranged to devote the upper part of the house to sick officers, the lower part to serious cases of non-commissioned officers, and to divide the marquees into three sections—surgical, medical and enteric.

On August 8th my duties as temporary acting military commandant ended with the arrival of Surgeon-Major Kilkelly, who had just been set free from the Portland Hospital. We were living in anxious times, and in spite of very hard work preparing the hospital we heard what was going on around us. We were told of Colonel Helyar's murder and how his English servant escaped and came into the British camp, quite incoherent with terror; the second police officer of our ward was shot in the arm and captured by Boers six miles from where we were; a big plot was discovered to assassinate the superior staff officers at Pretoria in their houses, to kidnap Lord Roberts and carry him off as a hostage to Louis Botha. The troops were to be absent by hurrying them to two big fires which were to break out in the west of the town, whilst the officers were to be attacked on the east.

Soon after the British troops entered Pretoria on June 5th the Governor formed the Pretoria Medical Commission, which commandeered everything likely to be useful for hospitals, and any hospital which applied could get a share of any stores in their possession.

There were several other hospitals in Pretoria, the Irish hospital in the Halls of Justice, the Burke hospital, originally intended for Boers, and which was later closed as dirty and unsatisfactory, and three model school hospitals. Another of these model schools was a prison where some British were shut up, and whence Mr. Churchill escaped. There was a splendid map of the Orange Free State on the wall, drawn by the officers, and a skeleton pointing to it, and some caricatures of Lord Roberts kicking out Krüger, which had been partially effaced by the Boers. It was from this school that the officers used to signal to a girls' school opposite. The girls succeeded in mastering the art of signalling, and then informed the officers of all the events of the war. Lord Roberts heard of this when he entered the town, and offered to give the girls tea at his house or go to the school. They chose the latter, and he went.

On August 18th our hospital was formally opened by Lord and Lady Roberts, and on the morning of the 19th we received our first patient, a yeoman with fever, followed next day by two officers, one suffering from the effects of sunstroke, the other, a Canadian, who had been transferred from the Burke Hospital. Late that night there arrived a string of fifteen ox-waggons, containing ninety-nine sick and wounded men from the battle-field of Commando Nek, under the care of a civilian surgeon. The convoy had been two long days on the road, and had camped on the open veld the intervening night. The oxen looked terribly exhausted and some had died *en route*. We had soup ready for the patients, and they were all put safely to bed with very little confusion considering that the experience was as yet a novel one for most of our sisters and orderlies. Their clothing was piled up outside the tents till next morning. Two surgical cases, a septic bullet wound of knee and an abscess of tonsils, had to be carried to the operating theatre that night for urgent relief. The cases known to be enteric fever were at once carried on stretchers to the enteric ward. One of them was partly unconscious and delirious and spent the night in trying to get out of bed. Many of the other cases were able to walk from the waggons to the tents and even attempted to carry their kit and rifles. It was very pathetic to see the patience of the sufferers, only to be matched by that of the silent, weary oxen. The next morning the invalids were all found to be doing well, and were full of gratitude for the comforts surrounding them. One of my patients,

however, who had not been labelled enteric, was evidently suffering from that disease, so I had him at once transferred on a stretcher to an enteric marquee, where the poor fellow died of hæmorrhage a week later, being the only death from this convoy. After this the hospital rapidly filled and more beds were opened, till eventually the total accommodation reached 560 fully equipped beds; 5227 officers and men were treated as in-patients, besides 1095 out-patients. To lessen the monotony of hospital life various forms of amusement and recreation were eventually organised, the band played, concerts were given, and even private theatricals were arranged by the staff. Golf links, hockey and football grounds were laid out in front of the hospital. By request of the authorities the hospital was kept open till September, 1901, when it was taken over by the army and became No. 22 General Hospital. It is to be noted that this is the first war in which civil voluntary hospitals have assisted the medical service of our army in the field.

Ten other hospitals under civilian surgeons did splendid work during the war, but time does not allow me to mention them. Those of you who want more information in a handy form will do well to read Sir Frederick Treves' little book, 'The Tale of a Field Hospital,' which has lately been republished.

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March 3rd, 1913.

CLINICALIA.

Extracted from the Clinical Journal.

By F. W. LANGRIDGE, M.R.C.S., L.R.C.P.

(*Continued from p.* 288.)

(160)

Mistakes not always the result of Ignorance.

"Even Homer sometimes nods," and the most alert of us sometimes become blind to the significance of symptoms and signs with which we were perfectly familiar at an earlier period of our professional career. Mistakes may thus arise, not so much from a lack of knowledge, as from the fact that the possibility of such and such a condition has "never once crossed the mind."

Such an obvious truism is submitted with the utmost diffidence and respect, as much as a reminder to one's self as to his audience, since, whether engaged in consulting or general practice, we all, even in this democratic age, live in palaces of glass, where it is unwise to throw stones.

("Interesting Cases of Abdominal Surgery, including some rare forms of Intestinal Obstruction," by Henry Curtis, B.S., F.R.C.S., 'Clin. Journ.,' vol. xxxv, p. 22.)

(161)

Colitis

and

The "Wild Races" of Bacteria.

In this group, bacterial colitis, the attacking organism is contained within the intestine. It may be one of the normal and ordinarily peaceful flora of the bowel, but in the graver cases it is probably one of the "wild races" which are often introduced with food and drink (especially by fly-contamination), from unclean mouths and teeth, and possibly from swallowed nasal and bronchial secretion.

("Treatment of Colitis," by Herbert P. Hawkins, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxv, p. 33.)

(162)

Nervous Colitis,

or

The Patient who is a Misery to Herself and others—especially others.

Finally there is a class (of colitis) in which nervous phenomena, either local (secretory, motor,

or sensory) or general, or both local and general, are a prominent feature of the disease. . . . The general phenomena mainly coincide with neurasthenia, by which term is meant a quick fatigue, little reserve and slow repair. The term "neurasthenic" does not, however, cover the whole ground. There is often added that state which greatly increases the difficulty of treatment, viz. a tendency to exaggerate small symptoms, a fancifulness, combined with hopeless obstinacy, and a weakening of the normal healthy desire to get well. Such a mental state is never present in the purely bacterial forms of colitis already described, and it stands out in strong contrast. Moreover, in this group alone the question of sex comes in, there being a great preponderance of cases in the female.

(*Ibid.*)

(163)

It is the Bird and not the Cage that counts.

A visit to the Salpêtrière, practically the same to-day as it was in Charcot's time, emphasises the fact that it is the man and not the building which makes a medical school famous, or, as Professor Babinski aptly put it, "the bird, and not the cage, is the all-important consideration."

("Medicine on the Continent," by Harry Campbell, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 53.)

(164)

Attitude of the Profession towards a New Theory.

Commenting on the theory that asthma is due to infection of the respiratory passages, Dr. Batty Shaw says: "Contemporary medicine, however, is not yet enamoured of this view, though there are signs that it would coquette with the notion."

("Asthma," by H. Batty Shaw, M.D., F.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 86.)

(165)

The Function of the Pituitary Body.

We do not know very much about the pituitary body. By one school in our student days we were taught to regard it much as the appendix is by some still confidently, but certainly mistakenly, regarded, namely as a useless and somewhat troublesome heritage from our simian ancestors. By another we were warned that, as the material envelope of the soul, we should be cautious, reverential, and not unduly curious in the attitude which we adopted towards it. In our own day both these views have happily been discredited, for it has been demonstrated that the pituitary gland has an internal secretion which is essential to the economy.

("Pituitary Extract and some of its Effects," by Leonard Williams, M.D., M.R.C.P., 'Clin. Journ.,' vol. xxxvi, p. 92.)

(166)

Laparotomy, the Operation of the Day.

Every surgeon, as a matter of course, and many a general practitioner, as a matter either of necessity or of vanity, now opens the abdomen.

(*Ibid.*)

(167)

The Victory over Intestinal Paresis.

It is therefore hardly too much to say that the post-operative terror of intestinal paresis need no longer beset the mind of him who, whether justifiably or otherwise, goes down to the peritoneum in rubber gloves.

(*Ibid.*)

(168)

Limitations of Iodides in Treatment of Syphilis.

I shall not dwell at any length on treatment by the iodides, which are of the greatest value in getting rid of certain syphilitic manifestations, though they do not appear to be capable of eliminating the poison of syphilis in the slightest degree.

("Syphilis: A Discussion," Mr. Ernest Lane 'Clin. Journ.,' vol. xxxvi, p. 181.)

(169)

The Beginning of Wisdom
and
Pasteur's Fine Thought.

In conclusion we may add with Professor Fournier of Paris, in imitation of another proverb, "that the fear of syphilis is the beginning of wisdom," and re-echo that fine thought of Pasteur's, "that where good is to be done duty only ceases when we no longer have power to achieve more or to do better."

(*Ibid.*, Major French, 'Clin. Journ.,' vol. xxxvi, p. 188.)

March 3rd, 1913.

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LECTURE ON ANGINA PECTORIS.*

By R. O. MOON, M.D., F.R.C.P.,

Physician to the Hospital for Diseases of the Heart.

FROM the time of the first description of angina pectoris by Heberden in the middle of the eighteenth century this disease, or rather, symptom-complex, has always exercised a considerable fascination over the imagination of the medical profession. The startling suddenness of its onset, perhaps for the first time amid apparently good health, the alarming and extremely painful sensations, with the possibility of a fatal termination always before one, all combine to make a vivid picture, comparable in its rapidity of onset and shortness of duration with epilepsy; in fact, this so much impressed the French physician Trousseau that he spoke of the disease as an epileptiform neuralgia. Now angina pectoris in its classical form is not a common disease, in fact, it is a somewhat rare one, and seen more frequently in private than in hospital practice. My own impression is that true angina has changed, or is in process of changing its type, and that the classical form of the disease with the sudden appalling breast pang, the sense of constriction and impending death is becoming much less frequent, and that we have in its place minor manifestations or "*formes frustes*" of the true angina, doubtless resting on the same pathological basis but presenting less definite clinical forms. Somewhat similarly the old-fashioned sthenic attacks of gout, which used to be called "the red-hot hissing gout," have become rarer, perhaps owing to the more abstemious habits of the population, while minor manifestations of it are only too common. So, too, in the case of angina we have an increasing number of anginiform attacks, but fewer cases of the classical angina.

Now the main features of an attack of true angina

* Delivered at the Dreadnought Hospital.

are well known to you, but we will just briefly go through them.

A patient in apparently good health is seized with alarming suddenness by a most severe attack of pain in the lower part of the sternum. Usually it occurs for the first time after some muscular effort: it may be a long walk, mounting a staircase, or lifting something heavy. A patient of mine says he always gets an attack if he goes out into the air soon after a meal, particularly on damp days. The attack may sometimes, however, occur without apparent provocation, *i. e.* in the middle of the night, when the patient is wakened out of sleep, but this is very rare in the case of the first attack, which is nearly always preceded by some definite muscular exertion.

The severity of the pain is very great, often described as agonising; the heart seems to be squeezed tightly in a vice, or the lower part of the sternum feels as though it were being pressed against the spine. A patient of mine describes his pain as usually beginning in the back; I have seen him in an attack when he localises the pain at the level of the fourth dorsal vertebra, and says he feels as though the chest and back were being squeezed together.

A characteristic feature of the pain is its irradiation; most commonly it passes down the left arm into the forearm mainly along the ulnar side, reaching to the fourth and fifth fingers, but not infrequently it also passes down the right arm; in these cases it is the brachial plexus which is affected, but the radiations of pain may pass into the region of the cervical plexus, reaching the neck, chin, lower jaw, and lobule of the ear.

The intellectual faculties remain clear throughout the attack, but there is a strange sense of impending dissolution which may cause the patient indescribable anguish, so that the psychical suffering, if we may so express it, far exceeds the physical pain.

The face grows pale, the extremities become cold, and the body is covered with sweat.

The condition of the heart and pulse usually remains unaltered; in some cases the beats are quickened. There may be a rise in the blood-pressure, but what is still more striking is the fact that the respiration is usually unaffected; there is no dyspnœa, but the breath may be held lest breathing should intensify the pain, or it may be shallow and quick.

The duration of the attack is very short; it may be a few seconds or one to two minutes, and then there is a return to comparatively normal health after the attack. Such seizures may recur at varying intervals, and in one of them a fatal syncope may supervene. Instances have been quoted where a succession of attacks have come on very close together, called by the French physicians, "*un état de mal angineux*," which is analogous to the *status epilepticus*.

Apart from these severe seizures of the classical type as sketched out above, other minor manifestations may occur which still own the same pathology and the same unfavourable prognosis.

(1) There may be a simple sensation of substernal constriction without any irradiation of pain.

(2) Pain in one arm accompanied by severe præcordial oppression.

(3) The attacks may be constituted solely by painful sensations situated at the periphery, in the arm or leg, without any pain in the chest.

(4) The agonising pain may be localised exclusively at the level of the epigastrium, accompanied by gastric troubles, *i. e.* vomiting, nausea, etc.

Pathology.—There is generally found after death some narrowing of the coronary arteries, which may be affected at their orifices or in any part of their course; there may be an atheromatous or a calcareous thickening of the arterial walls. This condition of the coronary vessels is a result of subacute or chronic aortitis, which may be caused by lues, gout, lead poisoning, or alcohol. I have been much impressed by the great predominance of lues in connection with true angina; in fact, since the introduction of the Wassermann reaction I have seldom met with a case of angina which did not give a positive reaction. The result of this damage to the coronary arteries is to impair the blood-irrigation of the myocardium, which, like the grey matter of the central nervous system, shows a very high degree of sensitiveness to an impaired supply of oxygen. This impairment of the vascular condition of the myocardium may be brought about by vascular crises in the coronary arterial distribution due to vaso-motor influences which are not associated with anatomical lesions of the arteries. This, of course, is exceptional, as patients who suffer from angina nearly always have arterio-sclerosis, either general or localised, at the root of the aorta, together with the changes we have men-

tioned in the coronary arteries with the consequent ischæmia of the myocardium.

It cannot, however, be said that this condition of sclerosis is the sole factor in the production of angina, for cases with sclerosed coronary arteries are by no means uncommon which present no symptoms of angina.

Ætiology.—The most common cause is over-exertion, the first attack occurring after some unwonted amount of exercise in a man past the meridian of life. Often the history is given of walking uphill against a wind when the attack first seizes the patient. Severe and agonising as the pain of this first anginal attack may be it usually passes off rapidly as the patient stands still, which he instinctively does, and thus gradually wends his way homewards very slowly and with frequent stoppings. After angina has once begun then quite slight exertion may often induce an attack, such as undressing for examination by the physician, as I have several times witnessed at the out-patient department. Not infrequently it occurs at night, possibly due to some flatulent dyspepsia, or as a result of a rise in the blood-pressure caused by the recumbent position. Occasionally the attacks come on at night at the termination of a dream, or, rather, a nightmare. Other slight causes which seem to provoke attacks are quick eating, an attack of coughing, yawning, sneezing, some rapid movement, some keen emotion such as anger, joy, or fear; and people who suffer from angina should be warned against the danger of flying into a passion. Sometimes the attacks are announced by prodromata, such as flatulence, meteorism, yawnings, or painful sensations in the limbs, which might be compared to the aura of epilepsy.

A further point to be observed in the ætiology is that angina is comparatively rare among working men and peasants, but more frequent among those who go in for good living and take but little exercise; it is also common among those who are exposed to cerebral overwork. For my own part I am disposed to think that worry and anxiety are important factors in giving rise to the vaso-motor spasm, which I believe to be the immediate antecedent of the great majority of attacks.

The most common age is between fifty and sixty, and it is far mose frequent among men than women. In contrast to this general rule, however, let me quote you the case of a young girl, aged nineteen, who was recently under my care at the Heart Hospital.

J. M—, æt. 19 years, had rheumatic fever at the age of eight, which left her with mitral disease. She was in the Waterloo Hospital in 1907 with a second attack of rheumatism, when her heart showed a harsh systolic and mid-diastolic murmur at the apex. I saw her five years later in 1912, when she came up to the heart hospital for very severe attacks of pain, anginal in character, which seized her suddenly at night, and were most severe in the left arm; the severity of the pain was such that they had been obliged to send for a doctor at night, who injected morphia. On admission the patient appeared somewhat cyanosed, there was considerable cardiac hypertrophy and dilatation, mitral regurgitation, and a soft blowing diastolic murmur along the left border of the sternum, with the second sound still audible there, indicating a moderate degree of aortic regurgitation. The anginal attacks at night continued to be severe, and the pain in the left arm was specially bad. On one occasion during the attack there was also great palpitation of the heart and the pulse-rate was 176, the forcible beating of the heart being audible a yard off. Nothing gave any relief but opium. However, after putting her on regular doses of iodide of potassium the attacks became less severe and finally ceased altogether. She was to have gone out of the hospital, but on the morning of her departure, just after eating her breakfast and apparently seeming well, she uttered an exclamation of pain and fell back dead. The post-mortem examination showed a large heart weighing 18 oz., the left auricle with calcareous plaques on the wall. The aortic valves were thickened and deformed and incompetent. The mitral valve was shortened and thickened. Such a case of anginal attacks occurring in so young a subject is, of course, quite exceptional.

Course and prognosis.—A single attack of angina pectoris must always carry with it a serious outlook. The first attack may indeed prove fatal, as in the classical instance of Dr. Arnold, and it is impossible to tell that any given attack may not have a fatal issue; in fact, sudden death is a common termination of angina. Still, with reasonable precautions a more hopeful view may be taken, and cases, such as the well-known one of John Hunter, have been recorded as lasting over twenty years, perhaps having but few attacks during that period. Here are notes of a case recently under my care:

R. D—, æt. 62 years, a cabinet-maker. The attack of angina came on suddenly while he was walking in November, 1911. Next attack occurred a week later, and when I first saw him a few weeks later they were becoming more frequent. Sometimes he would have two to three in a day; generally they came on when he was moving about.

On examination the apex-beat was in the fifth space ¾ in. external to the nipple line; the dulness showed some increase to the left, and there was a to-and-fro aortic murmur audible all over the præcordial area. The cardiac impulse was weak, and the blood-pressure rather low for his age, being 120. The Wassermann reaction was positive.

With rest and iodide of potassium (gr. viij *t.d.s.*) he improved considerably, and the attacks became quite infrequent, but I have recently heard that he died last November, just about a year since the occurrence of the first attack of angina.

Speaking generally, the more frequent and more severe the attacks the worse is the prognosis. When the seizures occur without any obvious provocation, and particularly at night when the patient is at rest, the prognosis is always grave, for the more easily the paroxysms are evoked the more extensive must be the coronary obstruction and consequently the more serious the complaint. During an attack a slow and irregular pulse is of evil omen, and so also is the *pulsus alternans*. One must remember that behind the symptom-complex of angina there is practically always an organic disease of the heart, and death comes more readily when there are cardiac complications. It is wise never to give a favourable prognosis even though you have failed to detect any organic disease of the heart; generally there will be found some morbid changes in the heart or aorta, or both.

Treatment.—We all know the efficacy of amyl nitrite in an attack of angina, best given in the form of glass capsules (♏iij–v), which may be broken and the vapour inhaled, the patient generally feeling most comfortable in a sitting or standing posture. Prior to the introduction of the nitrites by Sir Lauder Brunton it was customary to give injections of morphia, and this may be resorted to now with advantage sometimes, for opium has not merely a sedative but also a stimulating and vaso-dilator action, which renders it useful in these cases where nitrite of amyl has failed.

In the intervals the patient must be subjected to a fairly strict *régime*; in particular, alcohol and tobacco should be avoided, tea and coffee only taken in strict moderation and a generally light diet adopted. It is very important to avoid heavy meals, and rest after a meal should be strictly enjoined. We must always keep before us the necessity of reducing peripheral resistance where it is excessive. With regard to drugs I have found that iodide of potassium is certainly most generally useful, even when there is no history of lues, perhaps by reducing the pulse tension; the addition to this of liq. arsenicalis has seemed to me to moderate both the frequency and severity of the attacks. Other patients have experienced much relief from taking a mixture of liq. trinitrini ♏j and dilute hydrobromic acid ♏xv three times a day.

Angina Minor.

This is the form of angina which often goes under the unsatisfactory name of "pseudo-angina," and though it bears a superficial resemblance to the true angina it is in its essence a very different condition. Sometimes it is spoken of as angina vaso-motoria, but this seems to give rather too narrow a definition, for these conditions may not always be due to vaso-motor changes. As a matter of practice there is seldom any great difficulty in distinguishing these cases from those of the true angina. In the first place it is far more frequent in women than in men and in comparatively young women, whereas I pointed out that the most frequent age for true angina was between fifty and sixty. Then, again, true angina almost invariably makes its appearance for the first time after definite exertion, whereas this minor form often comes on for the first time when the patient is quietly resting. In several cases that have come before me there has been a definite history of some emotional disturbance, which seems quite clearly to have been the starting-point of these minor attacks. The seizure lasts much longer, perhaps two to three hours, and though the pain may be severe it is never so agonising as in true angina, nor is there the sensation of impending death. Patients may, however, become unconscious for a quite considerable length of time. As a rule there is no cardiac lesion, and certainly in such cases of this kind that I have seen the patients have seemed in other respects healthy, and I should have pronounced their hearts on physical examination to be organically sound. The ultimate prognosis as contrasted with true angina is nearly always good, though such cases are often obstinate to treat.

Coming in detail to the symptoms the pain tends to radiate centripetally rather than centrifugally as in true angina; thus the attack often begins with tinglings and shooting pains in the upper limbs, which then pass to the præcordial region; sometimes it appears as though the sympathetic were implicated. The pain is less fixed and more diffuse than in the severe angina; the sensation experienced by the patient is that of fulness, tension or weight in the stomach, the heart feels too big, but the feeling of its being grasped in a vice is quite exceptional. In an attack there may often be a certain amount of panting and slight dyspnœa. An important point of distinction between true angina and this minor form is that the latter may sometimes be relieved by movement: thus the patient jumps out of bed, walks a few steps and then experiences relief. These attacks often exhibit some periodicity; generally they occur suddenly when no effort is being made, or are preceded by slight troubles such as vomiting or nausea.

In specially nervous cases the attack may terminate with a sort of hysterical crisis, such as an outburst of tears or the passage of a large

quantity of clear urine. In the case of men this form is often associated with excessive tobacco-smoking, and is known as tobacco-angina; in this condition it appears that the nicotine causes a spasmodic stenosis of the coronary vessels.

Treatment.—During the attack it is well to apply heat to the trunk and extremities as the body surface is so often cold in these cases, ammonia should be given or other diffusible stimulant. If, as in true angina, there is arterial spasm, then, of course, amyl nitrite is indicated. When there is no such spasm, but the pain is severe, it may be necessary to administer morphia subcutaneously, but this requires some caution in a condition which is liable to become rather chronic.

In the intervals the important thing is to search for the possible cause of these attacks. The digestive system must be carefully supervised, as this form of angina is very readily caused by constipation or flatulence. The clothing, too, demands attention, so that too tight skirt-bands may not increase the dragging of the abdominal contents upon their supports. I have been much impressed by the frequency with which some emotional disturbance has given rise to these attacks, so that it is important to consider seriously the psychic state of the patient, and very often the best remedy will be a complete change of scene and circumstances for a few months; it does not much matter where it is, provided that no place of great elevation is selected, and that the conditions and society are congenial to the patient.

Of more direct remedies, the use of the artificial Nauheim baths seems to me to give good results. I had under my care at the hospital a woman, æt. 29 years, who was a school teacher. She had suffered from anginiform attacks for some years, which had first arisen in connection with an emotional upset. There was no organic lesion of the heart. After failing entirely to diminish the frequency or severity of the attacks while treating her as an out-patient, although she had abundant opportunities of resting at home I took her into the hospital, where she was treated with the artificial Nauheim baths, and for the last two and a half years she has been quite free from the attacks. More recently I have had a similar case in an older woman, æt. 55 years, who, however, has mitral regurgitation with some dilatation of the heart, and has on several occasions had epileptic fits. Her attack of angina minor began about nine years ago, the pain usually beginning in the left arm and then passing to the left side of the chest; often she had a shaking of the left arm and hand or a sense of numbness in that limb. With her the treatment has not been so strikingly successful; at the same time she has been considerably relieved by the baths, and she herself thinks they have done her more good than anything else.

March 10th, 1913.

PHARYNGEAL PHLEBITIS.

By W. STUART-LOW, F.R.C.S.,
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It is certainly remarkable that in all the records of pharyngeal affections no mention is to be found of phlebitis as a disturbing factor, not to say a veritable ailment; and this is even more surprising considering that veins are abundantly present in the walls of the pharyngeal cavity. Most of those veins are superficially situated and but slightly supported and covered by tissue, and are therefore easily susceptible to injury, and quite readily become inflamed. I have recognised and treated pharyngeal phlebitis as a definite, veritable entity in the category of pharyngeal diseases for over two years. In quite a large number of patients I am confident that the relief afforded has been due to treatment directed to this as the causative factor, and its elimination effected a cure which otherwise would have been long delayed or impossible.

Inflammation of the pharyngeal veins is a common condition, for about 30 per cent. of all patients who complain of persistently recurring throat-ache and discomfort in this region have had it as the cause. It is, therefore, important that such a common state of disturbance should be readily identified. Phlebitis is met with at all ages, but mostly in young females, especially if their mucous membranes are naturally thin and they happen to be engaged in work necessitating the breathing of a dry or dusty atmosphere.

In the normal state the arrangement of the vessels on the anterior surface of the soft palate has a horizontal course running inwards transversely from each side, except on the uvula, where the veins are vertical, running downwards to the tip. On the posterior surface of the palate the veins can also be seen with the retro-pharyngeal mirror to be horizontally arranged. On the posterior pharyngeal wall they are mostly vertical in direction, while at the sides they are obliquely inclined. In this position varicosity is sometimes most marked, and it is often a feature of the veins on the anterior palatal pillar, especially near its junction with the tongue. Tortuosity of these veins on the palatal pillars, the sides of the soft palate, and the pharynx

is also common. It is quite usual to observe a certain changed appearance of these vessels, consisting of enlargement and increased prominence—tortuosity and varicosity, and when these alterations have occurred, inflammation is more likely to supervene. There are, therefore, two predisposing conditions leading to the vessels of the pharyngeal wall becoming inflamed. First, undue thinness of the mucous membrane covering the vessels, which is normal to those who have inherited a thin mucous lining or inner skin; and secondly, shortage of the mucous secretion, which is the natural lubricant and protector. The walls of these vessels are normally thin, and this tends to favour varicosity and tortuosity, which thus constitute a predisposing condition to the ready development of phlebitis. Tortuous and varicose vessels are more prominent, standing out into the pharynx, and, therefore, more liable to friction by imperfectly masticated food, and to irritation by septic oral contents passing over them. These predisposing conditions may exist, however, and even be well marked, and if the oral cavity be normal, that is, aseptic, and the faucial tonsils healthy, often no phlebitis may supervene. One of the exciting causes in the onset of inflammation of these pharyngeal veins is oral sepsis from carious teeth or other dental conditions, inducing sepsis of the mouth and acting as starting-points for pharyngeal sepsis. Another is the condition of the tonsils, which are most frequently found to be enlarged and follicular, and, if so, are always septic. They may be quite small and embedded, covered over by the palatal pillars, but usually containing in their ramifying recesses much inspissated material which is very septic. Such septic retained secretion is constantly being passed out into the pharynx, and thus becomes an active cause of venous inflammation.

Pharyngeal phlebitis is most often met with in the lower classes, because the exciting conditions are more commonly present in them, viz. neglect of oral hygiene and inattention to diseased tonsils and adenoids. It is also not infrequent in the better classes, since hereditarily thin mucous membranes are often met with in them. This thin and dry condition of the mucous membrane of the pharynx in good class patients has usually been called gout, but it is most unlikely that it is gouty. Judging from my own experience, I am disposed to maintain that the inherited thin mucous membrane, which is very liable to become dry and glazed, and to have the veins on it inflamed, is not gouty, but is simply a result of what I have described as "hypomyxia pharyngea." It is a naturally thin mucous lining, and its invariable accompaniment is shortage of mucous secretion, and alteration of the character and quantity of the secretion, whereby it clogs and hangs about the pharynx instead of passing downwards and lubricating the œsophagus and supplementing the stomach action.

The duration of the symptoms that are chiefly complained of by those suffering from inflammation of the veins of the pharynx is generally considerable, unless they are brought to ask for relief by the acuteness of the pain during an exacerbation when the symptoms may have lasted only a few hours or days. In the more chronic cases the patients usually complain of an aching in the throat located in the posterior wall, or on one or other side, and sometimes in a more definite position, as at the junction of the palate and tongue, where the veins are mostly varicose and tortuous.

Again, patients describe the sensation felt in the throat as hot and burning, or throbbing, and sometimes a shooting pain at intervals. A sense of dryness is also characteristic, and a feeling as if something were in the throat, which is not surprising, as very often there are clumps or tags of semi-agglutinated mucus adhering to the walls. There is a sensitiveness to condiments, such as mustard and pepper, swallowing of which often starts a shooting pain. On inspection, many congested and often tortuous and varicose veins are to be seen. Where the vessels are largest, as on the anterior pillars of the palate and posterior wall and sides of the pharynx, these vessels often stand out prominently, and have a livid look. The mucous lining has a stretched appearance, and is usually more or less desiccated, and collections of mucus take place, and are to be seen on the pharyngeal surface. The patients complain of pain on swallowing, and are often thirsty subjects, and find drinking cold fluids cooling to their throats.

It is rare to find the typical pharyngeal phlebitis without a septic mouth from dental caries or septic gingivitis, tartar, or pyorrhœa alveolaris.** In the great majority the tonsils are enlarged and follicular, or embedded, retaining accumulated secretions in

their recesses. A dry, irritating, hacking cough is often complained of, or a tickling sensation is felt, and continues until it ends in a cough.

In the treatment of pharyngeal phlebitis it is, of course, essential first to have the mouth made hygienic; the dentist must be consulted to assist the throat specialist or practitioner in extracting all carious teeth and scaling away all accumulated tartar. If pyorrhœa alveolaris exists, it must be properly attended to. A menthol mouth-wash is an advantage both as an antiseptic and as a deodorant, for the breath is often very foul. "Dencol" (Duncan & Flockhart), which contains a small proportion of salicylic acid, is a very good and efficient preparation and a favourite with patients. A small quantity of quinine solution (one grain to one ounce of water) makes an effective mouth-wash. Tincture of myrrh is a very good preparation, as it hardens the spongy gums, and tends to fix loose teeth in their sockets; half an ounce of the tincture and eight ounces of water makes a useful, though not a very agreeable, mouth-wash. The teeth and mouth having been seen to, the tonsils must then receive attention. The best operation in all cases is enucleation, as after this has been properly performed a recurrence of hypertrophied tonsils is impossible. Sometimes, however, especially if the tonsils are pedunculated, the ordinary guillotine may be employed, and the whole tonsil can in this way be completely removed in such cases. Should these operations be refused, the next best procedure consists in slitting up all the tonsillar crypts and follicles and laying them all freely open, curetting out all the accumulated, septic, inspissated secretion, and rubbing the interstices up with a 25 per cent. solution of tinc. ferri mur. in glycerine. The painting ought to be continued daily, or every second day, for some weeks to harden, antisepticise, and to shrivel up the enlarged tonsils. The galvano-cautery point, to puncture the tonsillar tissue, has been employed for the same purpose, but I cannot recommend it; in fact, I condemn it as slow, painful, and ineffective, enucleation being necessary in the long run.

It has recently been proposed to revive the now obsolete cauterisation of enlarged tonsils; this is very surprising, since practical acquaintance with this method is enough to lead any competent judge to discard it.

Having got rid of the sources of the sepsis in the mouth and tonsils, the next step in the treatment should be to soothe the inflamed vessels and mucous membrane generally. One of the best ways of doing so consists in painting or spraying the throat with elixoid mucin, which is a preparation first introduced by me, made by Burroughs, Wellcome & Co., and is a solution of animal mucin, quite pleasant to use, having no odour and a sweet taste. It is the most ready cleanser of the mucous membrane that I have ever seen, one application being quite sufficient to clear off all accumulations of old secretion that so frequently clog the lining of the throat. It also nourishes the mucous membrane, making it thicker, moister, more resisting, and stronger. As in most of the patients affected with pharyngeal phlebitis, the mucous membrane being naturally thin, dry, and glazed (hypomyxic), such action of the mucin elixoid is most beneficial, and the application should therefore be continued for some time after the phlebitis has passed away, as a preventive of future attacks. Besides painting the throat with this preparation, I often order it to be sipped at intervals in place of lozenges, the dose being a dessert-spoonful, which should be poured into a glass and sipped slowly. When the more acute pain has disappeared, more astringent remedies may be used to contract the enlarged vessels and tone up the mucous membranes: equal parts of glycerine boracis and glycerine acid tannic act well as such a throat paint, or tinct. ferri mur. in glycerine, commencing with a 10 per cent. strength and passing on to 20, 30, and 50 per cent. If the patient can be taught and trusted to paint his own throat, it is well to allow him to do so twice a day, and always at bedtime, as the paint remains so much longer on the throat if applied on his retiring to rest for the night. I usually paint the pharynx with a solution of argyrol in glycerine 25 per cent., as this is soothing and astringent, but it is too expensive to give the patient to use as a paint. Another agent to be used by the medical man himself is a solution of chloride of zinc; the strength being 10 gr. to the ounce to commence with, which may be increased to 20, 30, and even 60 gr. It is a powerful astringent, but has the great drawback of having a very disagreeable nauseous taste. Where much throat painting has to be carried on by the patient, it is advisable to use a throat-mount carrying sterilised absorbent

cotton-wool, packets of which containing rolled wool are to be purchased. The wool can in this way be securely fixed, so that there is no danger of its becoming detached in the pharynx, and loose hairs from a throat brush are avoided. In many cases where the inflamed vessels are large and tortuous from repeated attacks of pharyngeal phlebitis, it may be necessary to destroy the vessels; this is best and most quickly done by the galvano-cautery point, the cautery being brought to a dull red heat and the point applied in a linear direction to the dilated vessels, one application being as a rule quite sufficient to destroy each vessel.

Patients with the thin pharyngeal mucous lining, and, therefore, likely to suffer at any time from pharyngeal phlebitis, should not be allowed to smoke, as the hot tobacco-smoke has on them a very irritating and detrimental effect, since it hastens the drying up of the throat, and a dry mucous membrane is always a vulnerable one. The mucous secretion is always deficient in such people, and therefore excess of salt in the food should be forbidden, as common salt has the power of dissolving mucus. If it is introduced into the food in any large quantity, the mucus, which is the natural lubricant and protector, is removed, and so the vessels are exposed to irritation and injury. Where the mucous membrane of the pharynx is normal, viz. thick and velvety, with a sufficient mucous secretion, smoking and the use of common salt are never likely to do any harm, but in those who possess what I have termed a hypomyxic throat lining their effect is harmful; it is these people who cannot stand the inhalation of tobacco-smoke and in whom smoking must be interdicted if they are not to drift into the category of the throat valetudinarian, always complaining, never relieved, and a trouble to all associated with them.

March 10th, 1913.

Safety Razor Blade Scalpel.—J. B. Murphy states that every surgeon has been annoyed by a a dull scalpel when a sharp one was wanted. Further, after a few strokes of the scalpel, especially in a firm or resistant tissue, like an old scar, the cutting edge becomes dulled. Sharpening scalpels is a nuisance and at best unsatisfactory. To overcome these objections, he has devised handles to carry safety razor blades. After these blades are no longer serviceable for shaving, they make excellent scalpels. They are cheap and easily obtainable.—*Journ. A.M.A.*

THREE FURTHER LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
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Lecture II.

The Care of the Sick and Wounded in Naval Warfare and the Russo-Japanese War, 1904–5.

In the previous lectures we have only considered the care of the sick and wounded of the land forces in time of war, and we have tried to realise the special difficulties attending the prevention and treatment of disease among soldiers. For several generations war has come to mean for us the meeting of armies in the field, and our navy, of which we are all so justly proud, has stood for a defender of our peace rather than as an aggressive power. But other nations have not all been so fortunate, and no one can foretell how soon the navy of England may be called upon, not only to guard the Empire, but to fight in its defence.

Within a period of ten years, Japan, whose wonderful reserve of strength and efficiency was, until lately, undreamt of by us Westerners, was engaged in two great naval wars, the war with China in 1894 and her yet greater encounter with Russia in 1904. Her manner of conducting these wars was a striking example of the genius of her people in adapting the best of Western progress to her own use. The war with China was fought just forty years after the appearance of Admiral Perry's fleet in Japanese waters, which first awoke that country from the condition of isolated stagnation which had lasted for more than two centuries. Of those forty years, fifteen had been spent in civil strife before the more progressive section of the community prevailed against the old order, and with the accession of the late Emperor in 1868 friendly intercourse with foreign nations was frankly accepted, and Japan began her splendid work of systematically and scientifically reorganising her political, naval and military forces. The standard

* Gresham Lectures.

of excellence to which she attained can be judged by the condition her forces were in at the outbreak of hostilities and by the result she achieved.

She had no tradition of naval warfare behind her, she fought without experienced veterans and without even the practical teaching which foreign naval wars could have given her, for there had been no great naval engagement fought with modern battleships, but she proved the value of her preparedness and of her minute attention to details. In no department did she prove herself more efficient than in her management of the wounded and of the naval hospitals and in the stamping out of disease among the crews. The medical history of Trafalgar and Lissa (1866) and of the naval war between Chili and Peru were of little avail, but when war broke out between Japan and China in June, 1894, elaborate preparations for the care of the wounded had already been planned and were at once set on foot. As soon as the stress of work occasioned by the big battle of the Yellow Sea in September of that year was over, a circular was issued by the naval department of the Japanese Government to the surgeons in charge of ships, naval barracks and hospitals, asking for detailed replies to an elaborate set of questions as to wounds, diseases and general sanitary conditions during the war, and from these replies surgical and medical reports were compiled by the Director-General of Medicine of the Japanese Navy, from which I shall quote to you.

The experience thus gained by the medical department of the navy stood them in good stead when the great naval war with Russia was fought and won ten years later.

In land warfare the wounds are generally caused by bullets or pieces of bursting shells; the victim falls and is carried to the rear by stretcher bearers. In only exceptional cases is such damage done to his surroundings as to hamper the movements of bearers, surgeons and dressers. In modern naval battles there are no bullet-wounds, but the damage done by a single well-aimed shell is terrific, carrying death and destruction with it. The report is full of accounts such as the following: "At 2.34 a 26 cm. shell perforated the mess-room on the port side of the lower deck, where the surgeons were stationed, destroying more than half the medicinal articles; then the shell perforated the torpedo magazine and, passing beneath the torpedo discharger, crushed the side plate at the entrance to the engine-room. Lastly, it struck against the gun-mounting of the 32 centimetres and tore it to pieces, without exploding, damaging stores, oil tank and medicine chests. By the scattering of the fragments of the iron plate, the beams above the torpedo discharger, the rails for the torpedo, a fish torpedo, the scullery, lavatory, bath room, part of the gun room and the surrounding parts of the lower deck were destroyed." Imagine the havoc after a dozen such shells had done their work in a single ship! The officers and men are either killed or injured by fragments of shells, by explosions and burns, or by splinters from planks or iron rails. There is no possibility, of course, of moving the wounded out of the danger zone during the course of the battle; all the surgeons can do is to choose a site for the hospital as handy as possible for the reception of the wounded and yet below the armoured deck. In the old sailing ships, such as the "Victory," there was a wide hatch extending from the upper deck to the very bottom, below the water line and in the centre of the ship. Here were space and comparative safety for a hospital, but in a modern battleship this space is given over to the engines, coal bunkers, etc., and the wounded must go elsewhere. As it is inconvenient and sometimes impossible to carry the wounded the whole length of the ship, the surgeries in the Japanese navy were placed, if possible, at either end of the ship; but this has its disadvantages because the surgeons and dressers and surgical instruments must then be divided, and it is obviously more convenient to have them together. In many cases surgeons must be content to utilise any space which is available, though that space may not be ideal as regards the perfect cleanliness modern surgery demands, and cannot even be safe from shell fire. Here is part of the report of Surgeon Kusano of what happened on board the man-of-war "Matsushima" during the engagement in the Yalu Sea: "About fifty minutes past noon the battle commenced, the bombardment on both sides growing gradually more and more violent, and a furious rain of shells from the enemy fell on all sides. About 2.34 I went out to give instructions for carrying the wounded! I crossed the deck to the starboard gun-room just opposite the temporary hospital, and at this instant a rumbling noise and a violent shock suddenly arose, and I

knew that some shells had struck our vessel; going back immediately to the hospital I discovered that the spot exactly where I had been standing a moment before had been obliquely perforated by a 26 cm. shell. The greater part of the surgical articles were destroyed by this shell, and the rest were scattered here and there. As work in the hospital under such circumstances was impossible I tried to prepare a hospital in a gun-room on the starboard side, but the majority of medicines and surgical instruments having been either damaged or lost, I thought it better to transfer myself to the hospital on the upper deck. . . . . About 3.30, as I was occupied in assisting Chief Surgeon Ogisawa, two 30 cm. shells struck the ship; one of them exploded with tremendous force against the shield of gun No. 4 . . . where the temporary hospital was stationed; the deck was torn up and the gas produced by the explosion filled all around so that nothing could be seen, and one could only hear an indescribable cry and tremendous sound. Two severely wounded persons who were just under treatment in the hospital died from the shock. I was momentarily stunned and had a peculiar feeling as if the ship was sinking. Thinking that all the officers would be standing on the after-deck according to their ranks, waiting an honourable death, I determined to die with them, but was nearly suffocated while endeavouring to reach the deck. . . . When I reached the back part of the officers' kitchen the clouding smoke cleared off and I discovered that our ship was navigating as usual, but a great many persons with burns came to the officers' room, some walking, some being helped by others. As these burns were severe the cries and groans were pitiable." Compare the mental and physical suffering of these wounded with those who fall on the battlefield, who, even if they have to wait some hours, can generally, in civilised warfare, be protected from further damage and be taken to the comparative peace and safety of the field hospital. No Red Cross flag can be hoisted over the ship's surgery to protect it from shells, and the surgeons and dressers run almost as great personal risks as the combatants. Thus, in that same battle, the ship "Hiyei," which bore the brunt of the fighting, not only had her surgery completely destroyed by a shell, but every member of her medical staff was either killed or severely wounded. In their place three able seamen were appointed to attend to thirty-seven injured, and no doubt did all they could, but the medical stores had been destroyed and for twenty-four hours the wounds received hardly any attention, till it was decided to steer for temporary anchorage and signal for medical assistance. When this arrived only the most necessary help could be given owing to the confused state of the ship, the absence of surgical appliances, and the hurry in attending to so many injured at the same time. The wounds were cleansed, fragments of shell or wood were removed, splints were applied where necessary and wounds were dressed with aseptic bandages. The worst cases were then transferred to transports for conveyance to naval hospitals. At the actual moment of battle, one surgeon assures us, men are so excited and elated that, with very few exceptions, no anæsthetic is needed in the most painful process of probing a wound or setting a damaged limb; the suffering is far greater later on when the excitement has subsided and only the anguish and the shock remain.

A difficult question on board ship is the conveyance of the wounded. It stands to reason that, where it is possible, a wounded man is transported best at full length on a stretcher, but in a ship there are steep ladders and narrow gangways to manœuvre, where no stretcher can be used, and the Japanese found that hands alone were best to carry the men and caused least delay, though in the case of extensive burns or fractured limbs this method often added greatly to the suffering of the patient.

The wounds of a naval engagement are usually more severe and are surgically less satisfactory than wounds received in land battles, because, as I said, we have not the small perforations made by bullets to deal with, but the larger injuries caused by fragments of shell, or iron or wooden splinters, or scalds and burns. These need far more careful tending and cleansing than the small flesh-wounds, and it is always difficult and often impossible for the surgeon to give them the attention they need at once. He has to deal with his patients in a surgery often by the dim light of an oil lamp, if the electric lighting has been damaged, where the air is filled with dust and dirt caused by the engagement. Under such circumstances it is practically impossible to keep the wound aseptic, and it must be remembered that one surgeon usually has to deal almost single-handed with many cases, all

needing immediate attention and many in awful pain. All he can do is to give urgent and temporary relief by washing the inside of the wound, disinfecting the surrounding skin, extracting foreign bodies where they can be easily seen and removed, staunching hæmorrhage and applying splints. When the battle is over further disinfection and examination must take place, though the utter exhaustion of surgeons and dressers often makes this a terrible strain. Not only are the conditions on board ship very unsatisfactory during warfare for the dressing of wounds, but shell wounds are very liable to suppuration owing to the difficulty of cleaning them, and many times, on more careful examination, pieces of clothing are found deeply embedded within the wound, causing inflammation and pain. Thus in Japan, in spite of all the skill the surgeons could give, the wounds did not heal satisfactorily, and it was found that the average stay of a patient in sick bay in the ship and later in a naval hospital was no less than two and a half months, which compares badly with soldiers wounded in land warfare.

I shall have occasion presently to speak of the comparative number of wounded and sick in different modern wars, but it is a striking fact that the cases of sickness in naval warfare are remarkably low compared with those in land forces. The sailor has not the same risks to run in this respect. The modern ships are sanitary, he does not gain access to improper food or drink, there is no hoarding of vast numbers together, nor has he to suffer from exposure and the short food supply which the soldier must occasionally endure, and above all he has a good supply of water to drink.

The number of deaths from disease during the war was only 141, a ratio of 9·88 per 1000 of the force, whilst those who died from the injuries received in action were 150, a ratio of 10·51 per 1000. I may briefly state here that, taking an average of recent wars fought on land, out of every 100 deaths, 20 die from wounds while 80 perish from disease. Perhaps the worst scourge of armies in the field is typhoid or enteric fever, which is due to infection carried in water or by flies. Only a few cases occurred during the naval war, which happy result was attributed to the minute attention paid by the medical and other officers to sanitary conditions, and to the obedience of the men in observing the precautions which were imposed upon them. Smallpox is endemic in China and Korea, but not a single case occurred among the Japanese naval men who went on shore on landing parties, although they mingled freely with the natives while the ships lay in harbour. This immunity was doubtless due to the strict enforcement of revaccination of all seamen which is carried out every five years in Japan.

There can be no better instance of the care bestowed by the Japanese on their fighting forces than the story of the practical eradication of beriberi in the navy. This disease, which is extremely prevalent in many tropical and sub-tropical climates, is now thought to be a so called deficiency disease arising from a diet wanting in some substance necessary to maintain health. It causes periphera neuritis and a consequent degeneration of the heart. It has been known since very early days, but no cure for it has been discovered. Modern research seems to have satisfactorily proved beriberi to be due to the absence of a substance called "vitamine" in the polished rice consumed by the natives of certain countries whose staple diet is rice. Where unpolished rice is eaten the disease does not occur. But to Baron Takaki, an old fellow-student of mine at St. Thomas's Hospital, the credit is due of proving, thirty years ago, that the disease could be stamped out by avoiding an excess of rice in the dietary of a Japanese sailor. He considered the cause to be an improper proportion of carbon to nitrogen in the food. In 1882, at the time he, as Vice Medical Director of the Japanese navy, was studying the dietary and evolving his theory, war between Japan and Korea seemed imminent, but most of the crews of the five largest ships in the navy were unfit for service in consequence of beriberi, and the question seriously arose as to whether the navy could supply a sufficient force for fighting. In the following year matters came to a climax when a training ship carrying 276 officers and men on a trip of 272 days developed 169 cases of beriberi with 25 deaths. On arrival at Honolulu, the proportion of proteids in the rations was increased, the rice ration was diminished, and no further cases occurred. A second ship was, at the instigation of Takaki, sent on an exactly similar voyage, but with rations arranged to contain a proportion of nitrogenous food considered suitable by him with a limited rice supply. In the course of a voyage of 287 days only 14 mild cases occurred, proving

satisfactorily that the disease could be checked by better chosen rations, and a reform was at once instituted, with the following magnificent results. In the year 1882 there had been 1929 cases of beriberi in the navy, in 1883, 1623, in 1884, the year the altered dietary was begun, the numbers fell to 718, and in the following year to 41, and since then, with a greatly increased force, there have been either no cases at all or only an isolated case recorded each year. In view of this extraordinary result it is surprising that the Japanese army authorities were slow to adopt the new method of feeding their men. At the beginning of the war with Russia, however, the number of cases in the army increased somewhat alarmingly, and the Minister of War ordered a mixture of barley and rice to be substituted for the rice ration alone, with an instant improvement and a diminished number of cases. The soldiers, like the sailors, did not like the alteration in their food, but submitted like reasonable men to what was ordered.

Before speaking further of the great war fought by the Japanese in 1904–5, it may be interesting to review very briefly the arrangements made in a modern British Dreadnought for dealing with the wounded in action. Two distributing stations are allotted for the casualties, placed behind armour and just below the water line, the principal one in charge of the P.M.O. and two sick-berth stewards. This station is permanently fitted with a hot-water supply and a ventilating fan; in time of war an operating table, an X-ray apparatus and several medical chests are added. The other station, for minor injuries, is in charge of the junior surgeon and remaining sick-berth steward. Great emphasis is laid, in the instructions issued to the surgeons, on the fact that only the most necessary operations should be performed in the dressing stations during an action, when the atmosphere must be highly unsatisfactory for dealing with wounds. As soon as possible after an action the wounded are to be transferred to a hospital ship, if available, or at least to a part of the ship where light and air can be obtained. The distributing stations are the headquarters for the stretcher parties, consisting of about sixty men, including ten dressers who have previously been trained in first aid and who carry bags containing dressings, splints and tourniquets, etc. When the bugle-call "Action" is sounded and the hands go to their war stations, the stretcher parties, consisting of non-fighting members of the crew, muster in a flat which is well protected and remain there during the whole action. When the firing has ceased during a lull in the battle the bugle sounds, and each party, under the command of one dresser, proceeds to a distributing station for orders, five parties to each station. They then proceed to search for the wounded in their allotted portion of the ship, working systematically from the upper deck downwards. When a dresser finds a wounded man he proceeds to dress him temporarily, then summons the four men under his command with a stretcher and they carry him down to the distributing station, where he is treated. The stretchers are all kept under armour and are located in convenient places, each dresser knowing exactly where to find his stretcher. The turret dressers remain always in their respective turrets, and two special small stretchers are provided for use in the turrets, where the hatch on the top is too small to allow one of ordinary size to pass through. A proportion of engine-room artificers and leading stokers receive special instruction which they can apply in action in the treatment of burns and scalds, and in the removal of the wounded from the coal-bunkers, stokeholds and engine-rooms. When the wounded have been treated they are secured to a plank and are put in racks usually used for stowing the men's bags, where the patients can be laid in comparative safety in rows three deep. Medical officers are warned, when an action is imminent, to transfer all medical stores, drugs, instruments, etc., to as protected a situation as possible.

The Japanese had fared badly in their land campaign against China in 1894, losing about three from disease to one killed by the legitimate casualties of warfare. But, taking the lesson to heart, they proceeded to mend matters, for the Japanese are nothing if not thorough. When their next great war broke out in 1904, and they fought and beat the Russians, they were not caught unprepared, and the net result of the lessons they had learnt and of their elaborate preparations was this: instead of a proportion of four dead from disease to one dead from wounds, which Sir T. Longmore tell us is the average in the wars of the last two hundred years, the Japanese reversed these figures, having a mortality of one dead from disease to four dead from wounds. By forethought, education and example, so many more thousands

of men, instead of lying as a heavy encumbrance in the hospitals, or of dying a miserable death, were preserved to fight for their country and bring honour upon their leaders.

In the Chinese war of 1894 the treatment of wounds had been antiseptic, but in 1904 a great advance was aimed at, for the wounds were treated aseptically and every effort was made to keep the wounds dry.

Reports bearing upon the medical services and sanitary conditions of the army in Manchuria in 1904 and 1905 have been made by British military and medical experts, who were allowed to accompany the troops, and the conclusion these authorities arrived at is principally that the Japanese relied upon the carrying out of ordinary sanitary measures necessary in war by every individual of each unit, rather than by any expert body responsible for such measures. The Japanese soldier learns from the moment he joins his regiment in time of peace that his health is a matter of paramount importance, not for his own sake, but that he may serve his country, and, if necessary, lay down his life fighting for her. A quiet unassuming patriotism is the religion of the Japanese, and when he has been made to understand how he can best help his country he can be relied upon absolutely to do so and to prevent becoming a burden upon her. Every man is determined to keep well from the highest motives; "the men know that they are fighting for the independence of their country," General Oshima told a British representative, "and that therefore they must do all they can to preserve their health in order that they may be of use."

In speaking of the exceptional absence of typhoid fever among the troops, General Burnett says: "I mentioned to General Nogi that although we had excellent sanitary rules and regulations for the guidance of the troops in the field, yet we had never succeeded in doing away with heavy casualties due to their non-observance. He replied that during the Japanese-Chinese war they themselves had suffered most severely in consequence of the ignorance or neglect of the men to recognise the necessity of strictly observing the sanitary rules issued for their guidance, more particularly amongst the younger soldiers, who were drafted to fill up gaps caused during the war. The authorities so took this to heart that, directly after the war, improved rules and regulations were drawn up, and all soldiers were thoroughly instructed in peace time as to their purport and the necessity of their strict observance and also what neglect of them meant to their country, army and comrades. This teaching had been so thorough and effective that the result sums up—practical immunity from all diseases which have hitherto followed the footsteps of all big armies in the field. . . . The old soldiers would take the younger ones in hand and insist upon their paying strict attention to rules which worked so well in their own cases."

A pamphlet is issued to every soldier, and is read to him and is explained at least twice every month. It deals first with instructions regarding the care of the body, and points out that even minor ailments, such as blistered feet, toothache and chilblains, impair the fighting efficiency of an army, and that many such complaints are aggravated by dirt. The soldier is enjoined never to neglect the daily washing of his entire body, of his teeth, and, as often as possible, of his clothes. Next, advice is given regarding clothing, which must not be too heavy, but must be warm, tears must be mended with an even stitch, or they will chafe, boots must be in good repair. Then follow directions regarding food, and more especially minute instructions about the dangers of drinking dirty water. Sufficient sleep must be taken before a march; the house where he is bivouacked must be clean or he must clean it, windows and tent flaps must be kept open, vermin must be guarded against with insect powder, and a special warning is issued about flies and mosquitoes. Precautionary measures are recommended against frost-bite, sunstroke and special infectious diseases. In this campaign a second pamphlet was issued to the soldiers about the measures to be taken against cholera and plague.

To the Japanese soldier there are three grades of illness in war time: first-class illness, which includes wounds or disease contracted by hardships and exposure, second-class illness, which comprises the ordinary diseases, and third-class illness, which is brought on by disobedience to orders, by immorality or carelessness, such as drinking dirty water or eating unwholesome food, and these diseases are a shame to the man who contracts them. "The people of Japan," remarked a distinguished officer, "do not like such soldiers, and they do not get the best of receptions if they leave the front in consequence of disease."

By means of this method of training a wonderfully high level of physical efficiency and mental development is attained in the army; the men are carefully chosen and kept well up to the mark. To the European soldier cleanliness seems a great difficulty when campaigning: but the Japanese is essentially clean, and contrives to keep it up in any weather and under any circumstances. "The Japanese soldiers always contrive to have a hot bath," says one reporter, "and on this subject no orders are necessary. Through long custom among even the poorest classes in Japan, a daily bath in water at very nearly boiling point is considered almost as great a necessity as food. On service the soldiers generally manage to bathe every other day, and even in the coldest weather a naked soldier standing in the open drying himself by the bath is a common sight. All through the winter this bathing was kept up. The large earthern jars about 4 feet by 2 feet which are to be found in every Chinese house are requisitioned for the purpose, charcoal or wood fires being lighted underneath." He brushed his teeth daily, for the Japanese as a nation take the greatest care of their teeth. Skilled dentists were appointed to the army who attended to all troubles of that kind. The commissioned officers of the Japanese army medical service consist of medical officers and apothecaries, and their status compares very favourably with that of the surgeons in the European and American forces. They have a very well defined position, their social rank is the same as that of other branches of the service, while they are proud of their profession—a profession highly honoured by men and officers alike. They have no executive authority, except over their subordinates, but they are the trusted advisers of the commanding officers in all matters concerning the health of the troops, and it would not occur to the military authorities to question their decision in such matters. The relationship between them and the combatant officers is an excellent one, and their advice is asked for constantly and is loyally accepted by officers and men.

Surgeon-Major Seaman, of the United States army, quotes this instructive little story:

"One hot day in July, 1904, after the battle of Kinchau, Oku's men were marching north through a region that abounded in the little gourd-like fruits which in that country are called water-melons. In one regiment the men who were attached to the medical corps, privates instructed especially to watch out for the health of the troops, reported that some of the men were suffering as a result of eating that fruit to slake their thirst. At once the regimental surgeon issued orders, or, strictly speaking, had them issued by the colonel, directing that under no circumstances were the men to eat any of the fruit. The heat was stifling, and the men were staggering along from exhaustion and thirst, but not one of them touched the fruit after these orders had been issued, although they could have done so easily; the physical disorder, which might have proved serious, was checked at once." Of course when any question such as the choice of camping ground or forced marching arose military exigencies were placed before medical advice, but friction between the medical and military authorities rarely, if ever, occurred. In nothing were the medical authorities more particular than on the subject of drinking-water. Time after time we have had this trouble with British troops, especially, perhaps, because their fighting has been chiefly in hot climates where thirst becomes overpowering, but if the Japanese soldier can be taught the terrible danger of drinking unclean water, surely we can give our men equal knowledge and show them the necessity of self-control.

In Manchuria the water supply was sometimes taken from streams which were often thick with mud, or, more often, from the well in the village street, a hole sunk in the ground with no coping to protect it from dirty surface water after rain. The Chinese village is one of the most filthy places conceivable, for the Chinaman is as dirty as the Japanese is clean. There was, of course, great danger in drinking water straight from such a source. After a regiment had been quartered in a village the medical officer made a chemical examination of the well and occasionally pronounced it unfit for drinking, in which case a notice-board was erected to state the fact, but where the examination was satisfactory a sentry was generally posted to guard it from contamination. The result would not have been so good, however, and we should have heard of more cases of water-borne diseases, if it were not the almost invariable habit of the Japanese soldier to drink boiled water. It is usually drunk in the shape of tea, for though the quantity of tea issued to the men was small they used it over and over again, till they were practi-

cally drinking hot water. A boiled-water station was placed along every route troops took in marching, any suitable vessel being used, and benches were placed for the men to sit and rest whilst they drank. Where the men could find no boiled water they used their own iron bottles to boil it, and these served the purpose very well. As soon as a regiment was established in a village the authorities set about building a coping round the well to protect it from impurities.

Where the Japanese was unable to boil his water he suffered just as others do. Thus, "there was, before Port Arthur, an advanced post of about 300 men, whose drinking-water came from a small well they themselves had made in the valley below their post. The post was in such close proximity to the enemy that fires were out of the question, boiling the water impossible. This water was good enough unboiled, until, one day in August, a heavy storm of rain came on which flooded the well and no doubt filled it with impurities. The men drank the water as usual, and next morning they were nearly all down with dysentery."

It would take too long to tell you in detail of the medical organisation of the Japanese army, and the facts would be too technical to be of general interest. The system has been based upon the field medical organisation of the German army, but the Japanese have adopted and simplified it to their own special needs, and the equipment of the mobile units resembles more nearly that of the British army. The conclusions Colonel Macpherson, of the Royal Army Medical Corps, came to (and he was present during the greater part of the campaign), were these: "There are one or two points which stand out prominently in connection with the Japanese military medical system. These may be briefly summarised as follows:

"(1) The extreme mobility of the field medical units, including the *depôts* for the supply of medical and surgical material.

"(2) The extreme simplicity and uniformity of the dressings, and the manner of dealing with the wounds.

"(3) The continuous and rapid system of evacuation of hospitals of all classes to the bases in Japan.

"(4) The method by which complete medical and surgical records are kept, even of those killed outright in the field, without unduly encroaching on the other professional work of the surgeons.

"(5) The general sufficiency of the Japanese medical service for all that it is called upon to perform under the conditions of modern warfare and big battles, and the ease with which the machinery works."

The only defect Colonel Macpherson points out in a system which is otherwise nearer perfection than anything we have achieved in Western warfare is a defect in the quality of the transport provided for sick and wounded, for the material generally consists of improvised stretchers, or rough country carts, goods waggons or trucks, without any arrangement for protection against cold, or for minimising the jolting which, in some serious cases, such as wounds of the skull and abdomen, causes unnecessary suffering and risk. "On the other hand," he says, "there is an entire absence of that sentiment which in Western nations is apt to exaggerate these defects into so-called medical scandals. The Japanese soldier who is wounded considers his sufferings as nothing compared with the hardships and dangers of his comrades who are left in the fighting line, and the general public appreciates this. The army staff recognises the defects, but so long as the ultimate result of the wound is not affected (and it is difficult to contend that it can be when a suitable dressing is well applied, and limbs, if necessary, immobilised), temporary suffering has to give way before the military exigencies, which do not permit of field units in the line of communication being hampered with special forms of transport material.

After speaking in such high praise of the organisation of the Japanese medical service, it is a somewhat ungracious task to criticise the methods of their opponents, but it may be useful to point out that the Russians fell into the time-honoured errors, which, campaign after campaign, have caused so much unnecessary suffering and such a waste of life among our own troops. In theory their army medical service provided sufficient *personnel* and stores for all the sick and wounded, but in practice it was supplemented by a very large staff of the Red Cross organisation, for the arrangements were made on much too small a scale for modern battles. In spite of the large amount of assistance given by five great divisions of the Red Cross Society, whose hospitals were far better and cleaner than those of the Army Medical Department, there was a deficiency of medical stores and

medicines and insufficient transport. The railway was largely employed in forwarding Red Cross sections to the front when such trains ought to have been carrying soldiers, and it was generally admitted that there was no sufficient co-operation between the Red Cross Society and the Army Medical Department, though both these organisations worked under the directorship of the chief sanitary inspector, who was, however, not a medical but a combatant officer. The army medical staff of the Russians had no administrative authority; they were used simply as doctors, the combatant officers having charge of all discipline. This led to divided authority and friction, and the results were unsatisfactory. The hospital arrangements made by the military authorities were very good, but they would have been totally inadequate for the numbers of sick and wounded if the Red Cross Society had not largely supplemented them. Practically all modern appliances were provided by this organisation. They established large stationary hospitals and formed mobile detachments which accompanied the troops. In many cases they did the work of the Army Medical Department and supplied the soldiers with small luxuries, such as tea, sugar and tobacco. The Red Cross doctors comprised among their number some of the most eminent surgeons and physicians of Russia, but unfortunately the great majority of the nurses were totally untrained women, whose zeal and devotion were unquestionable, but who could not rise to the demands made upon them in moments of emergency. It was, of course, the few trained women who were of real service.

After the great battle of Mukden the Russians left all their wounded on the field for the Japanese to care for—60,000 prisoners in all, including a large proportion of sick and wounded. It may be imagined what strain such numbers entailed on the Japanese hospitals. When Port Arthur fell 17,000 sick and wounded Russians were captured, the majority of whom were suffering from scurvy. The Russian soldier is of such good physique and is so well accustomed to hardships in his own home that he withstood the suffering caused by indifferent transport and extremes of heat and cold better than perhaps any other European soldier would have done. When the cold weather began there was no warm clothing provided, and the men, especially those in the trenches at night, felt the frost and bitter north wind terribly, till warm Chinese garments were mercifully given to them. But a great deal more illness occurred during the summer months, owing to the horribly insanitary condition of the camps, the foul water which was universally drunk, "which fault," a Russian officer remarked, "could not be remedied." The necessary rest for the men did not receive sufficient attention when such rest was essential to keep them in health. "Considering," Colonel Waters, of the Royal Artillery, says, "all that the forces under Stakelberg had gone through after June 13th, one might have thought they would be allowed to rest when possible. On the contrary, they were not. Kuropatkin inspected the 1st Siberian Corps at Kaiping on June 20th and distributed several decorations to officers and men. The former had a great supper in consequence, and kept a regimental band playing in bivouack from 9.15 p.m. till 11.30 p.m. An *aide-de-camp* to the Commander-in-Chief told me on that day that the health of the army had suffered very considerably, partly on account of exposure and exertion, and partly owing to polluted water and the utter absence of any sanitary arrangements whatever."

Several hundred cases of sunstroke occurred in the month of August in one army corps alone, which was not surprising considering the men were always fully equipped and only wore peaked forage caps on their heads. Of the 180,000 combatants under Kuropatkin there were, in the beginning of August, 15,500 sick and wounded, exclusive of those who had been evacuated to Russia. On August 12th it was stated at the Army headquarters that "the daily rate of sick was 100 for each of the first and fourth Siberian corps or 6000 a month for both of them," and this was at a time when no fighting was going on in these corps.

It is a pitiful story, but no new one. There is an incorrigible optimism among military authorities which, in spite of bitter lessons in the past, causes them to under-estimate the casualties of sick and wounded in war. But beyond that there is the want of proper instruction in sanitation which every officer, non-commissioned officer and every private of an army ought to possess. This lesson, above all others, we should take to heart from the Russo-Japanese war, that the health of the army and the fighting efficiency of the army are one and the same thing, and that this desirable end can only be achieved by the intelligent co-operation, not only of the medical officers and officers commanding regiments, but of all the non-commissioned officers and of all the men engaged.

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SOME RECENT ADVANCES IN THE TREATMENT OF SCOLIOSIS.*

By E. MUIRHEAD LITTLE, F.R.C.S.,
Senior Surgeon, Royal National Orthopædic Hospital.

SCOLIOSIS used to be better known, in this country at all events, as lateral curvature of the spine, but the former name has the advantages of being shorter and more descriptive and accurate. It has the small disadvantage of being Greek instead of English. The deformity is not uncommon. Hoffa and Dollinger found it accounted for 28 per cent. of all deformities, and Hoffa, in a collection of 2339 cases, found that 1974 were females and only 365 males—a proportion of over 5 to 1. Like many other facts this disproportion between the sexes has not been accounted for. (Nor has the disproportion in the sexual incidence of congenital dislocation of the hip.) It is not true for all forms of scoliosis or at all ages, but it is so for the most obvious and distressing form, that in which there is a double curve with structural changes and rotation. Some of the worst deformities, however, are found in rachitic boys.

Experiments on normal spines in the living subject are not very convincing, because if we exclude the cervical region the range of movement in the healthy spinal column is very small, except at one or two points, such as the dorsi-lumbar and lumbo-sacral junctions.

Therefore we cannot produce artificially anything like the deformity which occurs in scoliosis, no matter how mobile (within normal limits) the spine in question may be.

A great deal has been written on the causation of rotation, and the view most widely held now is that it is a normal constituent of the movement which occurs when a spine which is already curved

* Lecture delivered at the Polyclinic.

forwards or backwards is also bent sideways. Dr. Allis, of Philadelphia, has shown in a model that by altering the curvatures of the ribs rotation may be produced. The spinal column is so bound up with the ribs and rest of the thorax and with the abdominal muscles, that there is little true analogy between it and a flexible rod such as some observers have used to illustrate its behaviour. The action of the muscles is also to be reckoned with. Many of the muscles attached to the spine by one or both extremities must tend when acting unilaterally to twist the vertebræ. The fact that rotation accompanies flexion in cases of curvature from many different causes seems to indicate that it is an essential element in the mechanical changes which occur.

Advanced scoliosis is a complex deformity involving the whole structure of the thorax. Whether a case be due to congenital malformation, to paralysis, to rickets, to asymmetry of limbs, wryneck, empyema, etc., or, as in a majority of cases, to no definite known cause, the main anatomical features are the same, viz. a deviation of part of the spinal column to one side, generally, but not always, accompanied by a deviation of another part, or other parts of the column, to the opposite side.

This *deviation* is greater in the anterior than in the posterior part of the column. There is an angular displacement of each vertebra in the affected region, which is more or less rotated round a nearly vertical axis. From this "twist" the name "scoliosis" comes ("Σκολιός" = twisted). In other words, we have present that most troublesome factor of the deformity known as *rotation*. This rotation often accounts for more deformity than does the mere deviation, or it may constitute the whole deformity.

In a typical case the deformity of any individual thoracic segment consists in a folding in towards the spine of the rib on the side of the convexity, with great increase of curvature at and about its angle, and an unfolding or straightening out of the corresponding rib on the concave side, so that its curvature is much less sharp than the normal. At the same time the end of the transverse process on the convex side is approximated to the spinous process, while that on the concave side is bent away from it. Thus the one spinal groove is narrowed and the other widened.

Short of multiple osteotomy of the ribs, obviously there is no way of restoring the ribs to anything approaching normal form at one sitting. Therefore in a case with marked structural change cure by jackets must be a gradual process.

In congenital scoliosis, which generally depends upon the presence of wedge-shaped vertebral bodies, or other irregularity in form or in number of the elements of the spinal column, even if the deformity be corrected, the task of maintaining the correction is an extremely difficult one and implies the use of some retaining apparatus indefinitely.

It is very desirable to have a means of measuring and recording the deformity, for it is obvious that without some record it will be hard to say whether improvement has taken place. Much ingenuity and money has been expended in devising scoliosometers, some of which are very elaborate machines, such, for instance, as that of Schultess, but the practical difficulty has been due to the fact that accurate measurements of small changes of position of bony points which are covered by thick skin and subcutaneous tissues are difficult to make, especially when the bony points are rounded and not sharply defined.

Many, if not most, orthopædic surgeons prefer photography under carefully regulated conditions as the most useful means of record.

In addition to this I have found the following measurements helpful.

(1) *Deviation.*—This is measured in the erect position by dropping a plumb-line from the middle of the spinous process of the seventh cervical—the vertebra prominens—to the cleft of the nates, in which the line is allowed to rest.

The maximum horizontal distance from the cord to the spinous process in the middle of each curve is then measured and recorded. At the same time the point where the cord crosses the line of spinous processes is noted.

(2) *Rotation.*—The patient is laid prone and as straight as possible upon a horizontal board, with the arms by the side, and the chin, chest, hips and knees resting on the board.

A closed two-foot rule with an angle-plate at the joint and a spirit-level on one side is laid edgeways across the back, just below the lower angles of the scapulæ, with the spirit-level uppermost and the joint on the side of the prominent ribs. The rule

is then unfolded until the bubble in the spirit is central.

The rule is then lifted off and the angle read upon the plate and recorded.

The rule which I use was bought at a tool-shop for 1*s.* 9*d.*, and only required to have the spirit-level shifted.

It is objected to this method that the pressure of the table against the front of the chest tends to alter the deformity. This may be so to a slight extent, but I have found it, if a rough method, a very useful one.

It is advisable thus to measure the rotation first with the chest in full expiration, and again in full inspiration, and to record both results.

Scoliosis has long been a reproach to surgery, the treatment of any but slight cases, or those in a very early stage, having been generally unsuccessful as far as cure goes.

In the seventeenth century the deformity was treated by rivetting up the patient in a steel cuirass and back-piece, somewhat like defensive armour. An improvement upon this was the introduction of steel spinal supports, which are still used and have their value. Some surgeons and some quacks relied upon rest and recumbency on a prone couch, together with extension by weights, etc.

Others discarded all supports, and claimed great results from gymnastic treatment and massage.

Corsets of plaster-of-Paris and other plastic materials have been largely used, combined with suspension of the patient, and sometimes with forcible correction by means of traction or pressure by powerful machines (Redard, Barwell, Schultess, etc.). By these means lateral deviation could be corrected, but the rotation which causes the worst deformity generally remained little improved. In all these proceedings extension of the spine was aimed at, because it seemed obvious that to correct curvature it was necessary to stretch out the spinal column.

In the early days of subcutaneous surgery muscles and tendons conneoted with the spine were divided in a hope of facilitating correction. In 1903, Dr. Hoke, of Atlanta, U.S.A., divided a number of ribs in a case of scoliosis and diminished the deformity, but his example has not been followed as far as I know. Whatever means have been employed, the results have not been very satisfactory.

The following statement of what may be hoped for is quoted from 'Lateral Curvature of the Spine,' by R. W. Lovett, of Boston, U.S.A., 1907, and may be taken as not an under-estimate of results obtainable:

"(1) Total scoliosis should be entirely and permanently cured by adequate treatment.

"(2) Structural scoliosis in young children when of moderate degree should be practically cured by *adequate* and *long-continued* treatment, but only by that. If severe it should be much improved by the same means, the prognosis in both classes being better in children with a long period of growth ahead than in adolescents.

"Structural curves in older children and adolescents when of moderate degree should be greatly improved by *adequate* and *long-continued* treatment, but cannot be wholly cured. Severe structural scoliosis under these conditions can be markedly improved.

"Scoliosis due to severe congenital defects of the vertebræ, scapulæ or thorax, to infantile paralysis or to empyema cannot be cured but can be improved."

The difficulties notoriously met with in treating scoliosis are due to the following facts: In order to straighten a simple curve, such as occurs in bow-leg, we must apply force at three points, and in two directions.

The forces applied at the two ends of the curve must act in the same direction, and the force applied at the middle of the curve in the opposite direction.

This is just how a stick is broken across the knee.

But we cannot take such almost direct hold on the spine as on the leg bones.

Putting the cervical region out of the question, it is only through the medium of elastic bodies, such as the ribs and the masses of abdominal and lumbar muscles, that we can attempt to get hold of the spine. Therefore even the correction of lateral deviation is difficult, and the pressure on the ribs may increase the very deformity of those bones which we wish to correct. The attempt to take hold of the column in several places so as to rotate one region one way and another the opposite way seemed hopeless.

In June, 1911,* Dr. E. G. Abbott, of Portland,

* 'New York Med. Journ.,' June 24th, 1911.

Maine, U.S.A., stated that "fixed lateral curvature of the spine yielded to treatment as easily as bow-legs or club-feet." He further stated "that lateral curvature is a simple deformity which can easily be created artificially, as easily corrected and again established in the opposite direction with very little disturbance to the patient." Later, he wrote, "further experience has led me to believe that this deformity yields far more readily than either of the others."* These statements were supported by experiments with plaster jackets made on Mr. Stanley, a medical student who volunteered to undergo the somewhat unpleasant and very tedious experience.

"To effect a cure," Dr. Abbott says, "the spine must be flexed and drawn to one side by force exerted in the direction opposite to that of the fixed curve, and that, together with this, the low shoulder and depressed ribs must be elevated and forced into a position *posterior* to that of the high shoulder and bulging ribs, *i. e.* the patient must be placed in a position which is exactly opposite to that of the original deformity, *and this with the spine flexed* (italics are mine, E. M. L.). By this method not only is the spine over-corrected, but the integral parts of the deformity pass through the same arc which they traversed when the deformity developed."

To attain the desired end, Dr. Abbott places his patient supine upon a narrow hammock, which is stretched upon an oblong iron frame which rests upon four legs, which legs are connected by horizontal bars near the ground, thus forming a second, lower frame. This hammock can be stretched or slackened by a winch arrangement at one end of the frame, and was originally so arranged that its side corresponding to the concave side of the curvature of the patient's spine hung much looser than the other side.

The patient having been well padded over prominent bony points and covered with a vest, a large thick pad of wool or felt—the material is of no importance—is placed over the postero-lateral aspect of the concavity of the spine and secured with a turn or two of bandage. She is then placed upon her back on the hammock, and her legs are held up by attachment of the feet to a superstructure. Various bands of webbing or other strong material are fastened round the trunk at such points as seem necessary to correct the lateral deviation and to fix the pelvis, and tightened as much as seems necessary or prudent, and their ends fixed to the upper and lower bars of the frame. The hammock is slackened so as to allow as much flexion as possible, and—which is most important—the hand of the concave side is drawn forwards and upwards, and that of the convex side is drawn backwards and downwards, the shoulders and head being thus rotated in a direction tending to untwist the spine.

A strong plaster jacket, including the correcting bands and hammock, is then applied in the usual way, and made specially strong underneath and behind the raised shoulder, and above and in front of the depressed one. It is cut high at the bottom in front and low behind. As soon as the plaster has nearly set a large window is cut over the pad before-mentioned, and the pad removed through it. A second window may be cut in front on the opposite side over the flattened ribs. These windows leave unsupported parts of the trunk, and favour expansion of the concave parts in the direction of least resistance. The patient is taught to make frequent efforts to fully expand the chest, and so force the thoracic walls to bulge into the window or windows in the jacket.

Such, roughly, is Dr. Abbott's method. Since his last paper in the 'New York Medical Journal' appeared, he has been so kind as to send me a number of photographs which show that he has modified his apparatus, and some of the details of the treatment. Chief among these is the disuse of the oblique hammock with one slack side. The frame has now a much larger and more solid superstructure, to which the feet are attached and to which correcting bands may be affixed. There are also a number of ratchets in various positions by which the correcting bands may be tightened at will. He now cuts extra slits in the plaster in front through which felt pads can be introduced or removed as required.

It is not likely that one jacket will produce complete correction of the deformity unless the case is a very early and slight one. It may be necessary to remove the jacket and apply a new one at intervals. One of my cases has had four jackets in six months.

You will have noticed that so far only one

* *Ibid.*, April 27th, 1912.

curve has been attended to—the dorsal one. In the case of total scoliosis, as a long single curve is called, of course, this leaves nothing to be desired, but when, as is generally the case, there are two mutually compensating curves—a dorsal, or dorso-lumbar, and a lumbar—the latter must be at first neglected, except that a pad may be applied and a window made over the lumbar concavity.

The dorsal deformity is so much the most unsightly and important in its effects upon the contents of the thorax, that if we can correct that, the lumbar deformity as a rule calls for little attention.

When, however, the lumbar deformity, as sometimes, but rarely, happens, is the most obvious, it must be chiefly attacked.

When Dr. Abbott's first paper appeared I fear that it did not attract the attention that it deserved —at any rate not in this country. He was not known to us as an orthopædic surgeon, as many of his United States colleagues are known, and for us the paper lacked authority and evidence of successful application of his theories. But when the second paper appeared, in April of last year, with photographic and other records of a number of illustrative cases, it became evident that here was an innovation worth investigation. Accordingly I at once had an apparatus made according to Dr. Abbott's published directions, and endeavoured to carry out his treatment.

I have now had enough experience of it to be able to say that it is, at least in my opinion, a very valuable innovation, and that I have already been able to achieve more definite improvement, amounting sometimes to practical cure, than with any other treatment which I have tried. If—as it has happened—I have not succeeded in curing cases of severe structural deformity, such as some of those figured by Dr. Abbott in his paper, it by no means follows that the fault is in the method. I have not yet tried it in many such, and in those I have found some technical difficulties, which further experience with improved apparatus will, I hope, overcome. By Dr. Abbott's kindness I have just received a specimen of his latest apparatus, with which I hope to obtain still better results.

It is too soon to tabulate cases treated and results obtained, but I wish to bring before your notice the record of one case which I consider is now complete.

Case of I. E—.—Ivy E—, a girl, æt. 12 years, attended at the Orthopædic Hospital on June 28th, 1912. She had a marked scoliosis with right dorsal convexity and prominence of the right ribs at the back, together with a hollow right loin and some fulness on the left side of the lumbar spinous processes. The rotation measured with the rule as described amounted to fifteen degrees. On July 6th a jacket was applied on the frame on Abbott's principles with a pad and window on the left side. A few days later it was noted that the left dorsal region seemed to be coming into the window. The jacket was changed on August 7th, October 11th, and November 7th. On the last two occasions the frame was not used, but the patient was put into the desired position, seated on a stool. On November 7th the rotation was measured, and found to amount to only two degrees. The jacket was finally removed on *January 25th*, 1913, when no measurable rotation was found in the dorsal region, but there remained some fulness in the left lumbar region. She was ordered a course of massage and manipulations and exercises in order to restore the muscular tone. The left shoulder, which was elevated in the jacket, was still held high two days after removing it.

Cases of the grade of I. E— (Eden) have, perhaps, been cured by other methods, but only when the patient could be continuously treated for one or more years in an institution, or by devoting all her time to treatment in a comfortable home. For hospital patients such treatment has been out of the question. With the Abbott jacket hospital out-patients can be treated easily.

This patient, during the last six months, has had four gypsum jackets applied, and no other treatment whatever. She has gone about and amused herself and might have attended school.

As Dr. Abbott himself has remarked, the desired position can be attained without a frame, but only with difficulty, and it is far more fatiguing to the patient to undergo the rather lengthy process of applying a jacket in the erect or sitting position, which position, however, I adopted in applying the last two jackets of Ivy E—. On the whole, experience shows that the horizontal position on the frame is the best, as the patient can be fixed better, and the correcting bands applied with more accuracy and effect.

Whatever may be the final verdict of the profession as to the extent to which Dr. Abbott's method will cure severe scoliosis, there can, I feel sure, be little doubt but that he has made at one bound a very great advance—an advance, in my opinion, greater than any other made in the treatment of this deformity for many years, and one which redounds to the credit of his powers of observation and his perspicacity.

March 17th, 1913.

A CLINICAL DEMONSTRATION OF CASES OF CANCER OF THE TONGUE.*

By CECIL ROWNTREE, F.R.C.S.,
Assistant Surgeon, Cancer Hospital.

EACH of these two patients has been admitted to the Cancer Hospital for a large ulcerated growth upon the tongue, and you will notice that in many respects there is a great similarity between them. This is, however, one point of difference of overwhelming importance, and that is, that while in one case the cervical glands are enlarged, hard, and obviously infiltrated, in the other there is nothing of the sort apparent. You will observe that while in one case the growth is exuberant and papilliform, in the other the growth is smoother, and more deeply involves the muscular substances of the tongue. It is interesting to note that it is in the second case that the glands are affected—a fact which bears out one's experience that the projecting or villous type of epithelial cancer anywhere in the body is in general of a less malignant nature than the forms in which ulceration and infiltration are the main factors.

In connection with the differential diagnosis of these cases, I take it that when we meet with a large ulcerating mass infiltrating the tongue, there is not very much room for doubt. Still, there is always the possibility of confusing gumma and carcinoma, and I should just like to refer to the means we have of distinguishing them, apart from the ordinary methods of inspection and examination. The greatest assistance is now furnished by watching the results of an intra-venous injection of a full dose of neo-salvarsan, which almost invariably clears up gummata of the tongue with great rapidity—in fact, I know no other part of the body where improvement is so certain or so quick. The result is that within five or six days a definite diagnosis is usually possible, and this is, of course, a great advance upon the more tedious process of administering potassium iodide, and waiting two or three weeks for the result.

There is, however, a very important source of fallacy that complicates the use of salvarsan in the differential diagnosis of these tongue conditions, owing to the fact that in very many cases of cancer of the tongue the malignant process is engrafted as it were upon a substratum of syphilis. The latter is, of course, influenced considerably by the salvarsan, and the general improvement in appearance may be so great as to be very misleading.

There must always remain a small residue of cases of such difficulty that it is impossible to give a firm opinion, at any rate within a reasonable time, without proceeding to the last resort of diagnosis—the removal of a portion of the tumour for histological examination. Here again, there are pitfalls, for unless the specimen removed be of fair size, and is deliberately taken from a suitable part of the growth, the result may be negative or indeterminate owing to degenerative change. A negative histological report has not, therefore, anything like the same finality as actual demonstration of cancer-cells.

Considerable weight is sometimes attached to the presence or absence of the Wassermann reaction—quite improperly I think, for although a negative reaction suggests that the condition under investigation is probably not of syphilitic nature, yet a positive reaction, on the other hand, does not furnish a particle of evidence that the affected tongue is not carcinomatous.

We should never allow ourselves to be unduly biassed by a previous history of syphilis; for I think it may be fairly said that any ulcer of the tongue of uncertain nature is very much more likely to be carcinoma than anything else. "When in doubt anticipate cancer" is a good rule in the surgery of the tongue.

These two men have only just been admitted to the hospital, and these methods of diagnosis have not yet been used; but granting for the moment that in each case carcinoma is present, what is to be our treatment? Can either of them be subjected to an operation which has for its object the complete eradication of the disease? I think that in the first case this should be attempted; in the second I believe it to be useless.

I do not propose to describe the steps of the various operations utilised for the removal of the tongue and the ablation of the glands, beyond emphasising the fact that whatever operation is chosen must be thorough: the whole tongue must be removed, and each side of the neck must be cleared of all lymphatic-bearing tissue. But I would rather refer to certain points that you will

* At the Polyclinic.

inevitably be asked about in any cases that you may have to deal with.

When told that the removal of the tongue is necessary the first question a patient asks is, "Shall I be able to speak?" and the second, "Will the growth return?" As regards the latter, I am afraid it must be confessed that the results as regards recurrence, except in the very earliest cases, are most unhappy—sufficiently good to justify the extensive operations now practised, but still bad as compared with many other sites. But in considering this point it has to be remembered that in spite of radium or other forms of treatment, the disease when not submitted to operation has a mortality of nothing less than 100 per cent.

As regards the question of speech we are on firmer ground, and the patient may be fairly told that very satisfactory speech will remain. that it will improve every day, and that, unless there are special circumstances in the case, it will not be so defective as to seriously interfere with his livelihood. At the same time I think the patient should be told that his speech will certainly be no worse after operation than it would be in a few months' time if no operation were attempted.

A radical operation decided upon, there are certain important preliminaries to be attended to. Every detail of the after-treatment must be explained to the patient; he must be told not to attempt to speak after the operation, and he must practise writing on a slate whilst lying in bed. He should also practise feeding himself through a short rubber tube attached to the spout of a feeding cup. Needless to say, another important preliminary is scrupulous cleansing and scaling of the teeth by a competent dental surgeon.

Whether the operation shall be done at one sitting or in two stages is a matter that must be carefully considered. Minute dissection of both sides of the neck is a prolonged and tedious matter, which may take from an hour to an hour and a half, and I think it is usually best not to attempt all at one sitting. If the operation is to be done in two stages, should the tongue or should the glands be dealt with first? I believe it is better to do the glands first, for the reason that after the removal of the tongue a wound is left which is bound to become more or less infected. If the glands are removed while this infected wound is present in the mouth, prolonged and dangerous suppuration is quite possible. Another and a cogent reason for the removal of the glands first is that personally I can conceive no more terrible experience than the removal of one's tongue; and it is reasonable to suppose that many people, after having gone through that, would hesitate before again submitting themselves to the surgeon.

The next question to decide is the choice of an anæsthetic. Chloroform is of course most generally used, given by means of Junker's apparatus; but the administration of an anæsthetic in a tongue case, particularly if it is advanced, is always a difficult matter; and, moreover, the anæsthetist is always in the way of the surgeon. Many experiments have been made in order to get over this difficulty, but I will only refer to two. The intravenous administration of ether sounded very promising, but unfortunately the technical difficulties and the complicated apparatus have condemned it, at any rate for the present, and we at the Cancer Hospital have discontinued its use. Injection of ether into the muscles of the buttock gives a beautiful, steady anæsthesia, but unfortunately the after-results have been most unhappy, for several of the patients have been temporarily lamed as the result of the local reaction of the ether, and this method has also been dropped. For the present, therefore, chloroform is still the anæsthetic of choice. In order to diminish some of the difficulties of anæsthesia many surgeons perform a preliminary laryngotomy, but I think this is unnecessary as a routine, although it is of the greatest value in unusually difficult or troublesome cases. During the removal of the tongue very particular care must be taken that no blood be permitted to trickle down into the trachea, and it is also important that no blood should be swallowed, for the presence of blood in the stomach nearly always incites vomiting, and for a patient to vomit after this operation is a very undesirable thing.

As regards the after-treatment, the posture of the patient is of the utmost importance. He should be propped up in bed at the earliest possible moment, and if in two or three days' time he is well enough to get out of bed into a chair, so much the better. The mouth should be cleansed by irrigation with a dilute solution of sanitas or with peroxide of hydrogen, and here again it is useful to accustom the patient to this procedure before the operation.

I now want you to suppose that neither of these

cases is suitable for a radical operation. In that case, what is to be the fate of these men? What happens when carcinoma of the tongue is left to itself? At the present time neither of them is suffering particularly. They both have sore tongues, they both have a little difficulty in speaking, and they both have a little trouble in swallowing. But they will both get worse, and in a short time the tongue will become so fixed to the floor of the mouth that their speech will become more and more difficult to follow, their difficulty in swallowing will become greater, and consequently they will begin to have trouble in getting rid of the saliva, which is poured out so copiously in these cases. We see, then, that in two or three months' time their condition will be one of absolute misery, and it is very important to remember that their misery will depend to a much greater extent upon the condition of the tongue itself than upon the condition of the lymphatic glands or the floor of the mouth. This being so, I feel very strongly indeed that in many cases of cancer of the tongue, where, owing to the extent of the disease a radical operation is out of the question, yet it is justifiable to remove the tongue itself. We shall not cure the patient, but we shall relieve him of much of his discomfort. If all the growth cannot be removed, or if a recurrence takes place at the root of the tongue, it is surrounded by soft tissues, and the patient is very much more comfortable than if he had a painful ulcerated organ firmly fixed in the floor of the mouth and to the lower jaw. The risk to life of a simple Whitehead operation is inconsiderable, the period of convalescence is short, and the relief of pain and discomfort is very great. Experience can be our only guide in dealing with such cases, and I am convinced from what I have seen that these limited operations in suitable cases are very well worth doing. There are cases, of course, where it is quite out of the question, and there are others where it has been done and recurrence has taken place in the floor of the mouth or in the stump of the tongue. What can we do in these cases? What measures of relief are at our disposal? The most important symptoms are, I think, three in number: firstly, the horribly offensive condition of the mouth leading to chronic septic absorption; secondly, pain; and thirdly, salivation. The first step to take is to remove the teeth. This sounds a drastic measure, but I am sure you must have been struck by the difference in the local condition when cancer of the tongue occurs in the edentulous. I am afraid that extraction of the teeth is frequently neglected; and I think it is a pity, for it undoubtedly helps to relieve the pain by removing a source of irritation, it certainly diminishes the fœtor, and it facilitates the cleansing of the mouth. In any case the teeth are of no use to the patient, for by the time that the stage with which I am now dealing has been reached, solid food can no longer be taken.

The pain of advanced cancer of the tongue is due firstly to the tenderness of the ulcer itself, and this can be relieved by painting it with the various anæsthetic solutions made for the purpose. Secondly, it is due to the fixation of the intrinsic and extrinsic muscles, with the result that every act of swallowing and every attempt at speech pulls upon these infiltrated structures. Finally, the most severe pain is, of course, occasioned by the implication of the lingual nerve in the growth. This was formerly dealt with by the division of the nerve, but the results were not very satisfactory, and I believe the reason must be that by the time such a procedure becomes necessary, the growth has spread beyond the confines of the distribution of this nerve, and has involved other branches of the third division of the fifth. Much better results are obtained by destroying the latter at its exit from the foramen ovale by an injection of 15 to 20 minims of 80 per cent. alcohol directly into the nerve.

The constant salivation is a source of considerable trouble, for it interferes with the patient's sleep. It can be diminished, and very markedly diminished, by the simple expedient of administering tincture of belladonna in small doses of 3 to 5 minims at frequent and regular intervals. I do not think that belladonna is as yet much used for this purpose, but there is no question as to its efficacy in controlling this and other forms of secretion, and the relief afforded is very great.

Finally, gentlemen, I should like to emphasise my belief that cancer of the tongue is to a large extent a preventable disease. Were it not for the ravages of tertiary syphilis and the results of dental neglect, cancer of the tongue would be rarely met with. Every wart or fissure, every patch of leukoplakia or chronic ulcer should be regarded as a potential carcinoma and should be treated accordingly.

March 17th, 1913.

ON THE CAUSES AND AVOIDANCE OF ABDOMINAL RIGIDITY UNDER ANÆSTHESIA.*

By J. D. MORTIMER, M.B., F.R.C.S.,
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St. Peter's Hospital for Stone, etc.

THIS difficulty is, or should be, usually absent, or only present to a slight extent, but its results when it is marked may be serious, especially when the operation involves the same region. There is interference with respiration, and consequently with due intake and elimination of the anæsthetic; dilatation of the heart will follow; the surgeon is hampered by extrusion of the bowels, by venous oozing, and by the difficulty or impossibility of reaching the part at which he is aiming. His work is interrupted and prolonged, and his intentions may be to a great extent frustrated. There is increased probability of vomiting and other after-effects. Fatalities have even occurred as an indirect consequence.

Causation.—Rigidity is due to mechanical *tension* and to active *contraction* in proportions varying according to circumstances, and of course also to the muscular development and nervous sensitiveness. Reflex contractions are usually marked in neurotic people, in alcoholic subjects, in drug-takers, and in children, especially when rickety.

As is well known, the presence of *food or much fluid in the stomach* sets up persistent straining, and it may be difficult or impossible to prevent vomiting.

Faulty anæsthetisation, as in failure to choose an *anæsthetic appropriate* to the patient, operation, and position, in uneven or too hurried induction, in allowing a degree too light for the occasion, and so forth, may be expected to cause trouble when the adminstrator is inexperienced.

Local disease, such as the presence of acute appendicitis, or of a gastric ulcer, may set up rigidity of over-lying muscles persistent under anæsthesia.

The position of the patient.—Certain positions greatly facilitate the work of the surgeon, but tend to increase the trouble under consideration to an extent that is, I believe, not fully realised, and to which I desire to draw special attention. This can be well appreciated by anyone who will put himself on his side with a large round cushion underneath it, and in that position will try to breathe freely, or will put himself for a few minutes in the Trendelenberg position. Even the ordinary recumbent position with the patient at full length is not such as we prefer when we wish to relax the abdomen for diagnostic purposes, and tension is obviously increased by putting a cushion or sand-bag, as sometimes required, under the lower ribs or pelvis.

The Trendelenberg position not only causes some tension, but also disturbance of the respiration, by impeding the action of the diaphragm, upon which the abdominal contents are thrown, and by congestion of the medullary centre and upper air-passages, especially in fat and plethoric subjects, in whom it may, however, for surgical reasons be imperative.

Operative proceedings.—These, as was some years ago pointed out by Macewen, may set up spasmodic contraction, even under very deep anæsthesia.

Traction of the mesentery or broad ligaments, enucleation of an adherent prostate, injection of a bladder ulcerated from tuberculosis, are especially likely to cause trouble.

Hiccough may be set up by manipulation in the region of the diaphragm.

Prevention and remedies.—To render abdominal operations easier much may be done by those in previous charge of the case, if there is time and opportunity for improvement of the general condition, particularly in correction of states of obesity, plethora, alcoholism, and rickets.

Means, which need not be here detailed, should, when practicable, be taken to get the stomach empty and keep it empty, particularly in emergencies and when there is intestinal obstruction. Straining and vomiting when a patient is in position for an abdominal operation are not merely troublesome, but very dangerous from the risk of ejecta entering the air-passages. This has occurred even under spinal analgesia, and is, of course, still more likely to happen under general anæsthesia.

Faults of anæsthetisation such as those already mentioned must of course be avoided. In the supine position, if not inconvenient to the surgeon, both shoulders should be raised (the one from which the head is turned rather more than the other). In the lateral position the uppermost arm

* Read at the West London Medico-Chirurgical Society.

must be supported on a rest; the patient is steadied and breathing less hampered. Loin cushions are sometimes faulty as regards size and adaptability to the patient's contour, or may be in a wrong position, so that there is needless tension of structures and interference with breathing. In the Trendelenberg position the shoulders should always be supported, and the weight of the head taken off as much as possible by one or two pillows, which may sometimes with advantage rest on the anæsthetist's knees. If support is lacking rigidity is markedly increased.

Spinal analgesia usually abolishes the trouble, besides keeping the bowels still, and is especially useful for this purpose in some pelvic operations. It should, however, be combined in most cases with general anæsthesia, which may be light when it would otherwise need to be deep. I have known many instances of serious psychic shock and other interruptions to the operation, also prolonged mental disturbance afterwards, when spinal analgesia has been employed alone.

After *hypodermic injection* of omnopon or morphine and atropine I have often found absence of reflex rigidity in cases (for instance, hysterectomies) in which it might have been otherwise expected. There is, as a rule, a considerable saving of the anæsthetic. It is worth while to inject (if previously omitted) just after induction or even at the onset of rigidity, for an effect generally follows in about ten minutes. I think, however, there is more liability afterwards to intestinal atony, and consequently to nausea and other ill-effects dependent thereon. After-pain, and vomiting which may be associated with it, is however alleviated. It need hardly be said that the customary precautions must be taken in administering such drugs, and that the patients need careful watching after the conclusion of the operation until their effects have passed off.

Nitrous-oxide or ethyl-chloride, followed by ether from an inhaler, or even ether by the open method and preceded by atropine, will, in unsuitable subjects, cause laboured respiration, persistent even when a change to another anæsthetic is made after induction. Under prolonged administration of nitrous-oxide, or of nitrous-oxide and oxygen, rigidity is very liable to occur, as is admitted by those who advocate their use for abdominal operations. Chloroform cannot be relied upon when given in a low percentage, and in a higher one its dangers are much increased by the existence of irritation of vital centres, and of any degree of asphyxia; it is also when fully given apt to be followed by persistent after-vomiting. Still, it answers well for some patients, particularly in the Trendelenberg position, which lessens the probability of shock. Ether by the open method or a chloroform-ether mixture (according to the state of the patient and the stage of the operation) are usually more effectual, and can be pushed with less risk. With a more extended use of C.E. mixture instead of chloroform for prostatectomy I find that depression and rigidity can generally be avoided. Whatever method be employed, it is of first importance to attend to respiration, and if this becomes unsatisfactory, to discover why it is so, and apply the appropriate remedy. Oxygen is often valuable, but should not be used merely to counteract cyanosis dependent on causes otherwise remediable. For example, one should not give ether from a closed inhaler to a full-blooded elderly person, and then turn on oxygen to alleviate its bad effects, masking a symptom after the manner of the prescribing chemist. Reflex rigidity from surgical proceedings is accompanied by more or less spasmodic closure of the larynx and retraction of the tongue, and efforts must be made to maintain a free air-way by such means as pushing forward the lower jaw, keeping out the tongue, and introducing a tube between the tongue and palate. Non-aëration increases rigidity, the extraordinary respiratory muscles being called into action, so that a vicious circle is formed. The depth of anæsthesia must be cautiously increased. It may be necessary to ask the operator to desist for a few moments whilst these measures are being pursued. I need hardly remind you how surgical authorities have insisted on the need for minimising reflex disturbances by making an adequately long incision, by clamping and cutting adhesions when possible instead of tearing them, and by avoidance of rough handling.

It may be well to mention incidentally that in some patients, such as those who are the subjects of chronic bronchitis and emphysema, respiration habitually is mainly abdominal, and the excursions of the diaphragm and abdominal walls may be very troublesome to the operator, although there is not tonic contraction. For obvious reasons this trouble unfortunately cannot be abolished, although much may be done by proper selection and administration of the anæsthetic to reduce it to a minimum.

I venture to say, in conclusion, that on difficulty arising from rigidity the surgeon and anæsthetist do not always appreciate each other's position. Whilst witnessing operations I have sometimes been struck by the forbearance of the surgeon when rigidity, apparently avoidable by the anæsthetist, has occurred. On the other hand, I have known the anæsthetist blamed when it arose from causes beyond his control, and could not be abolished by him without risking a fatality.

March 17th, 1913.

THREE FURTHER LECTURES*

ON THE

RELIEF OF THE SICK AND WOUNDED IN WAR.

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic; Chairman of the County of London Branch of the British Red Cross Society.

Lecture III.

Red Cross Voluntary Aid.

The Geneva Convention has been in existence since 1864, and has certainly exercised an influence in warfare wholly to the good. Civilised nations are daily becoming more loth to settle their disputes by force of arms, but even the most convinced believers in a peace policy must feel doubtful whether war will cease to exist for many generations to come; therefore let us do our utmost to mitigate the suffering which follows in its train. It must always be remembered that no army medical service can, for financial reasons alone, be kept of such strength as to allow of almost indefinite expansion when the state to which it belongs enters upon a prolonged and bloody campaign. At such moments the service must turn to voluntary help, and experience shows that when patriotic enthusiasm is aroused the army medical authorities are inundated with offers of money, goods, and personal assistance. But such offers, unless they come through a duly authorised channel, are rather an embarrassment than a help, and to Dunant we owe the initiative of founding in every country of Europe, America and Japan, a properly organised Red Cross Society, which is the recognised channel for the administration of relief and the distribution of gifts in time of war.

"The ideal Red Cross Society," says Sir John Furley, "is a national institution which keeps only one object in view, and that is the alleviation of the sufferings of sick and wounded sailors and soldiers in war." But it should be the centre of various other philanthropic societies, each performing an important work in peace, and yet so constituted that, whilst training its members for the relief of victims of the many disasters which are of daily occurrence on sea and land in civil life, they can be depended on to give substantial aid to the Royal Army Medical Corps in times of war.

* Gresham Lectures.

Every nation avails itself in some degree of voluntary assistance to supplement its army medical service, and it must be owned that many States are far ahead of us in the perfection of such organisations. This arises from the fact that we are slow to adopt any scheme relating to military affairs, for we obdurately refuse to believe in the likelihood of England being involved in war, excepting always such smaller wars as we, as a great colonising power, must from time to time engage in with uncivilised races. For these small campaigns our Army Medical Service suffices, and on this conviction we are apt to rest content. But the magnitude and duration of the Boer war gave us a rude awakening from this comfortable doctrine, and convinced many of us of the necessity for greater activity in training in days of peace the voluntary assistance which so generously comes forward in times of national danger.

During the early part of the war, when enthusiasm was great, the hospitals in South Africa were flooded with gifts provided by a generous, but often unwise public at home, for many of the presents, such as alcoholic stimulants, were wholly unsuitable and could not be allowed. An order had to be passed that all gifts should be sent through the Red Cross Society, which did its utmost to forward them to the particular hospital for which they were intended. These presents, generally most acceptable and most gratefully welcomed, were ultimately received by the nursing sister or the quartermaster of the hospital, and were distributed under orders from the medical officers.

"To receive and distribute these gifts to everyone's satisfaction," says the official account, "was an impossible task, but much depends on the tact and common-sense of the officer in charge of the hospital. As long as wars are waged such gifts will be pressed for acceptance, and, no matter whether they be required or not, they must be received and accounted for." Lists of suitable and acceptable gifts should, in time of war, be announced by the Red Cross Society in the public press.

But the difficulty of dealing with unsuitable presents is nothing compared with the trouble of dealing with unsuitable or untrained personal service, and to cope with this difficulty it is the constant desire of the Red Cross Society to train such personal service to be ready when the need arises.

By the time the sixth Division for the war in South Africa had been equipped with a sufficient medical staff, the personnel of the Royal Army Medical Corps had been practically exhausted, and for the seventh, eighth, ninth, tenth and eleventh Divisions, besides the Mounted Infantry and Colonial Divisions, the personnel had to be found locally or from voluntary agencies at home.

There was from the first a deficiency in trained orderlies of the Royal Army Medical Corps, and an offer from the St. John Ambulance Association of men trained by them was accepted. Altogether about 1900 superintending officers and men of the brigade were employed at various times during the war, and, though the military medical authorities sometimes complained of their lack of sufficient technical training and their ignorance of hospital work, their assistance was invaluable. Besides the help obtained from England a volunteer ambulance corps was organised in South Africa to supply 2000 bearers to carry the wounded off the field and transport serious cases from the field hospitals to the base on stretchers, for this mode of transport is far the most satisfactory for a seriously wounded man. A motley crew of all kinds of men was collected, not smart to look at, and often the butt of much good-natured chaff from the troops, who gave them the name of "body-snatchers" and "pick-me-ups." But they improved as they got into the work and became a very reliable, handy body of men. "They did splendid service," wrote Sir Frederick Treves, "and one which was keenly appreciated. They were the means of saving many lives and an infinite amount of pain. Their longest tramp, of which I had knowledge, was . . . a distance of twenty-five miles. They showed the usual British indifference under fire and went without hesitancy wherever they were led. Unfortunately it happened that many of the worthy 'body-snatchers' were wounded and not a few of them were killed. They were popular with the soldiers. They had the gift of tongues of a kind, and could compete with most in the matter of lurid language. Their incessant hunger and indiscriminate thirst were a matter of admiration. They were good-hearted, and, although they looked wild, they meant well. Many a wounded man has been rocked to sleep on their stretchers, and on more than one dying ear the last sound that fell was the tramp of their untidy feet."

The insufficient number of trained orderlies proved to be an extra strain upon the Nursing Sisters, who could not feel proper confidence in inexperienced men, and additional sisters had to be recruited, partly from the Army Nursing Reserve, from colonial sisters, and from nurses locally engaged in South Africa. "There were also," writes Sir William Wilson, "many offers of service from ladies, some of whom were not qualified either technically or by education, but who were 'willing to learn.' Although wholly untrained women could not be employed, those partly trained were at first engaged, but were gradually eliminated as trained women took their places. . . . It is impossible to say too much," he continues, "of the services which the Nursing Sisters rendered in the South African war. Their devotion to duty, often under very trying conditions, was only exceeded by their kindness to their patients, many of whom owed their lives to the attention of these ladies. As regards the quality of their work, while the average was very good, there were, of course, variations, and the best sisters were undoubtedly those from the staffs of the large hospitals at home."

Speaking of the sisters who worked with him in the war, Sir Frederick Treves wrote: "They seemed oblivious to fatigue, to hunger, or to any need of sleep. Their ministrations to the wounded were invaluable and beyond all praise. They did a service during those distressful days which none but nurses could have rendered, and they set to all. . . an example of unselfishness, self-sacrifice and indefatigable devotion to duty. They brought to many of the wounded and the dying that comfort which men are little able to evolve, or are uncouth in bestowing, and which belongs especially to the tender, undefined and undefinable ministrations of women. The English soldier is as sensible of attention and as appreciative of sympathy and kindness as is any other man who is at the mercy of circumstances, and I can well believe that there are many soldiers—some of them now in England crippled for life—who will long keep green the memory of the sisters. . . . Some weeks after Colenso I was at Pietermaritzburg and was looking up in the hospital wards certain cases. Among them was a paralysed man to whom one of the nurses had been very kind at Chieveley. I found him comfortably bestowed, but he was possessed of a handkerchief, the extreme dirtiness of which led

me to suggest that, as he was now in a centre of luxury, he should ask for a clean one. To which he replied, 'I am not going to give up this one; I am afraid of losing it. The sister who looked after me at Chieveley gave it to me, and here is her name in the corner.'"

From the first it was obvious that the establishment of medical officers from the Royal Army Medical Corps would not suffice for the Field Force, and the help of civilian doctors was asked for. As time went on more and more of these were employed, till they formed a large proportion, at one time 54 per cent., of the staff of the general hospitals despatched from England. It was found that the assistance of local practitioners of country towns in South Africa, who volunteered for service in the army, was particularly welcome, as they were put in charge of small improvised hospitals on the lines of communication, and their influence among the Boers, who have considerable respect for the doctor, ensured better treatment of these isolated parties than they would have received if they had been in charge of a military official or a stranger.

The help given by the eminent consulting surgeons who proceeded to the war was gratefully acknowledged by the medical officers, but some of them were averse to the employment of physicians and sanitary experts, considering themselves quite as capable to deal with the very difficult questions of the treatment of medical cases and of the practical methods of camp sanitation.

This was the first occasion on which civil hospitals were employed to supplement the hospitals of the regular medical service of the British Army. "No one who served in the late war in South Africa," says Sir William Wilson, "fails to appreciate the services rendered by these units, services which were not only of the greatest value to the army generally, but also enabled the medical service, of which they formed a part, to complete the heaviest task that has ever fallen to its lot. Civil hospitals, such as those which helped us in South Africa, will always be welcomed by the Royal Army Medical Corps on any future occasion on which the patriotism of the people sends them forth." These hospitals, equipped and provided on a very handsome scale by private funds, were officially affiliated to the Red Cross Society, and as the war continued depôts of this Society were established at Cape Town, Durban and other places, and wherever the British soldier went the Red Cross aided him in some form or another throughout that vast area of operations.

The amount expended by the British Red Cross Society in the South African war was £162,296; twenty-one commissioners and agents were employed, grants in aid were given, clothing and medical and other stores were provided.

A hospital ship under the special patronage of the then Princess of Wales, Queen Alexandra, who personally supervised the arrangements, made several voyages from Cape Town to Southampton.

At the relief of Kimberley the Red Cross stores were the first to enter the town for the relief of the sick and wounded. In Natal the Princess Christian hospital train, sent from England, consisted of seven bogie carriages, four for seventy-two invalids and sixteen orderlies, and three for medical officers and two sisters and other members of the staff, a dining room, a dispensary, linen closet, kitchen, pantry and store-room. This train was the first to pass over the bridge crossing the Tugela in March, 1901, and to enter Ladysmith with waggons loaded for the garrison, which had been relieved three days earlier. Next morning it started on its return journey with ten sick officers and fifty-four soldiers for Durban, where they were embarked for England. It carried during the ensuing months 321 officers, 19 sisters and 7208 men. A little luxury which was provided for each invalid entrained, and which was immensely appreciated, was a kit bag, containing soap, brushes, a clean shirt and other toilet necessaries. To the invalid, often ragged and with little or no kit, such forethought seemed a blessing out of all proportion to the value of the gift which the kindness of sympathisers had provided.

From the reports of Dr. P. R. Lowe, the Civil Surgeon in charge of H.R.H. Princess Christian's hospital train, I cannot forbear making one short quotation, dated October 13th, 1900:

"Tommy Atkins never shows up better than in circumstances such as these. There he lay on the bare floor, tired out, hungry and cold, with clothes soaked and stiffened with blood that had oozed from wounds, bound round roughly with puttees or whatever came to hand, and all that night, as soon as he could be moved, the sisters and the staff of the train worked, and, taking each in turn, strove to wash off the stains and dirt of the battlefield and

get his wounds into something like a satisfactory state of surgical cleanliness—yet never a murmur, only patient waiting and a jest when his turn came, and some there were with a bullet through the abdomen, and knew the peril of it, and some with shattered arms and legs, and some shot through the chest, and one without an eye and the bridge of his nose—one or two, indeed, there were who were not so patient, but these were delirious, hard to be kept in bed, and had bullets through their brains. It was a busy night, and the cook was as busy as the rest, with his hot soup and other equally welcome comforts."

In every war since the adaptation of the Geneva Convention there have been complaints of the abuse of the badge of neutrality which the Red Cross confers; some of these accusations have been based on misunderstandings of the Convention, where the wording has been somewhat ambiguous, others have been obvious blunders, and others, unfortunately, have been deliberate and intentional abuse. Among the misunderstandings may be quoted the case of Surgeon Captain Longhurst, who was made a prisoner by the Boers at Lindley because the arms and ammunition of his patients were found, according to regulations, in his pack store. The Convention permits of the reception of the arms of the sick by field medical units, and it would be difficult to say what should otherwise become of them, but the Boers insisted on more than one occasion that the presence of arms constituted a breach of the Convention and confiscated the transport and other material, while the personnel were made prisoners. Again, Major Burtchaell, R.A.M.C., was taken prisoner while tending the wounded on the battlefield of Magersfontein, although he was proceeding under the Red Cross flag to the Boer lines. While he searched for wounded he left his horse tied up, and some Boers investigated his wallets, where they found an unloaded revolver. There is nothing in the Convention to prohibit this, but the Boer regulations did not allow a medical officer to carry arms, and he was taken prisoner. Perhaps the wording of the Convention should be made clearer, for mistakes are difficult to avoid.

Unintentional blunders occur even more often, and this has always been the case. The day after the battle of Inkerman (in the Crimean war), Russell was with some working parties who were burying the dead, a temporary truce having been agreed upon with the Russians for this purpose. To the indignation of the British there was reported shell firing from the Russian ships in the harbour, and Russell was nearly killed by a shell. Years after a Russian officer spoke of it, and said the firing had been decided on because it was obvious to the Russians that under pretence of burying the dead the British engineers had been seen choosing sites for new batteries. "I know your engineer officers wear cocked hats and I myself saw your men carrying fascines." But the cocked hats were only those worn by the officers superintending the burying parties, and the supposed fascines were the litters on which the dead were carried. These mistakes are very common, and give rise to many stories of treachery which each side is too ready to detect in the enemy.

Mr. J. B. Atkins writes that he "remembers an occasion at Willowgrange in the South African war when he overheard a sergeant and his men complain that the Boers were firing at them from under a white flag. It certainly seemed true. There were the Boers occasionally visible along a ridge across the valley, and on the face of that ridge flew a white flag. On examining the 'flag' carefully, however, the writer found that it was a dead grey horse, which seemed to tremble in the mirage. On another occasion, near Heilbron, he informed an artillery officer that he was firing on a Dutch ambulance train. In the distance and bad light the train looked exactly like an ordinary convoy, which the officer had taken it to be. As the officer looked again through his glasses a puff of wind blew out the Red Cross flag, and the writer can never forget the expression of profound concern and regret which came over his face as he recognised his mistake."

The many complaints which arose on both sides that the personnel and transport material of the medical corps were fired upon must chiefly have arisen from the fact that the Red Cross on the ambulance waggons and on the brassards worn by the men were not distinguishable at the modern long ranges.

Those cases where the abuse of the badge is intentional it is hard to forgive. Longmore, in his 'Manual of Ambulance Transport,' gives several instances. In 1870, 300 Irishmen landed at Havre as an Irish ambulance and then fought at the

defence of Chateaudun. Again, in the South African war, there are the notorious instances where the Red Cross was abused by neutral States, as in the case of the International Red Cross ambulance organised in Antwerp, and the American Red Cross ambulance organised in Chicago. Most of the members of these ambulances joined the organisations solely as a means to enter the Transvaal, where they afterwards joined the Boer forces as combatants. It was also said that the Boers made use of ambulance waggons to cover tactical situations; as at the battle of Brankenlaagte, where they were unable to move their guns until our ambulances went out, when the guns were taken away under cover of the waggons. But that a great scheme of charity should occasionally be abused does not really detract from its merits, and every campaign serves to raise the Red Cross mission in the eyes of those who watch its work and realise its influence.

The German Red Cross Society was founded immediately after the signing of the Geneva Convention, to carry its recommendations into effect. Men are trained to serve with the army as stretcher-bearers and attendants on the sick and wounded, and women are taught to nurse. Sick nursing was, in 1864, entirely confined in Germany to certain religious orders, and from these Red Cross female volunteers first received their training. There were at that time no women nurses in the military hospitals, but a few religious sisters were gradually introduced, and in 1897 the Prussian War Office first engaged some army sisters drawn from the Red Cross sisterhoods, so that there are at present about seventy of these employed. The Red Cross sisterhoods are at the disposal of the War Office in time of war; the sisters have to undergo one year's training in a hospital followed by the teaching of "moral, religious and patriotic principles," extending over another year or two years, and a short course of nursing every third year in hospital. The sisters correspond approximately to our army sisters, but their work is voluntary and is regarded more from a religious than a professional standpoint, which is the survival of the old tradition when nursing was confined to religious orders. In case of mobilisation, each sisterhood must, within ten days, supply the Central Committee of the Red Cross with at least half their number of trained nurses. They must also be prepared to serve their country in case of epidemics, floods or other disasters, and, if called upon to do so, they must be ready to serve a foreign state. The earnings of each nurse go to a common fund of the sisterhood, which provides for the nurse during working life and in sickness and old age. The Society consists of sisters, who are supplemented by auxiliary sisters and lady helpers, who receive a much modified scheme of training.

It is interesting now to reflect that we have arrived at the centenary of the year when the ladies of Frankfurt united to found the "Frauenverein" of that city. This was the first voluntary society founded in Germany, and perhaps in the world, to alleviate the sufferings of war, "as far as such work might be included in the sphere of woman's usefulness, and it wished especially to be employed wherever there was need of instantaneous relief."

Relief was indiscriminately afforded by the ladies to the volunteer soldiers of Frankfurt, to Spaniards when returning from captivity, and to French prisoners and sick who had been received into the hospitals of that city. After the campaign had passed into France the ladies occupied themselves with an epidemic of typhus which it had left behind.

The Japanese Red Cross Society is practically an army medical reserve, including doctors, nurses and entire hospital equipment, so that in time of war the Army Medical Service is set free from the military hospitals in Japan to go abroad, leaving the Red Cross Society in charge of the home stations. When the Japanese Government became a signatory to the Geneva Convention in 1886 the Society of Benevolence, instituted in 1877, changed its name and became the Red Cross Society of Japan, to organise efficient training of patriotic volunteers in times of peace in order that they might be of real value in assisting the Army Medical Service in war. The personnel, including doctors, apothecaries, nurses, etc., take a solemn oath on admission to hold themselves ready at any time during a fixed number of years to respond to the call for service in war or other public calamity. The oath covers a period of five years for most but in case of the female nurses, they contract for fifteen years. Red Cross members are only under very special circumstances allowed to work with the army at the front, and then only in relief corps. In time of peace there are no women

nurses in the military hospitals, and the Red Cross nurses are engaged as civil nurses throughout the country. When war breaks out they take the place of the military orderlies in the base or reserve hospitals. In the Russo-Japanese war the Japanese Red Cross Society carried out the following work: the Ladies' Committees prepared bandages and dressings; they were present at the railway stations to see the troops off and welcome them on their return; transport columns were kept ready for Manchuria; the nurses were employed in the reserve hospitals, and the male nurses were engaged there and in the garrison at Liao-tung. The Society has over one million members, and its organisation has become a part of the life of the nation. In 1904 the Society's income was a quarter of a million pounds, and a reserve of 3000 trained nurses was ready for immediate service.

The Red Cross Society of Italy gives a three years' training to its nurses, and the members have been busily engaged during the recent war with Turkey. H.R.H. the Duchess of Aosta was serving as a nurse of the Neapolitan Section on board the hospital ship "Menfi."

I hope, to-morrow, by means of lantern-slides, to illustrate the work of the British Red Cross Society in this country and in the present war in Europe.

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March 17th, 1913.

"Welcome" Brand Tuberculin Bouillon Filtrate, Human (T.O.A.); "Welcome" Brand Tuberculin Bouillon Filtrate, Bovine (P.T.O.) —Two new preparations have been added to the list of "Welcome" Brand Tuberculins issued by Burroughs Wellcome & Co. These are "Wellcome" Brand Tuberculin Bouillon Filtrate, Human (T.O.A.), and "Welcome" Brand Tuberculin Bouillon Filtrate, Bovine (P.T.O.). Each is issued in hermetically sealed phials of 1 c.c., both undiluted and in dilutions representing 0·0001 c.c., 0·001 c.c., 0·01 c.c. and 0·1 c.c of T.O.A. or P.T.O. The undiluted preparations are for the use of practitioners who prefer to make their own dilutions. The Bouillon Filtrates T.O.A. and P.T.O. were the earliest tuberculins prepared, though not the first introduced into therapeutics. Being the weakest of all the tuberculins, they are now employed by many workers to initiate a course of tuberculin treatment according to the intensive system of dosage. The dilutions of the "Wellcome" Brand preparations are suitable for this purpose. They are prepared at the "Wellcome" Physiological Research Laboratories under expert bacteriological control.

"Tabloid" Thyroid Gland gr. $\frac{1}{10}$ and gr. $\frac{1}{4}$. —In thyroid medication the dose of thyroid gland has usually been regarded as 3 to 10 gr., and is so given in the majority of books. Recently, however, certain authorities have stated that they have found doses of gr. $\frac{1}{10}$ to gr. 1 equally satisfactory. To provide for this new system of dosage two additional strengths—gr. $\frac{1}{10}$ and gr. $\frac{1}{4}$—of "Tabloid" Thyroid Gland have been issued by the same firm. "Tabloid" Thyroid Gland is standardised by chemical means so as to ensure that the desiccated gland substance, of which each product represents a definite amount, contains not less than 0·2 per cent. of iodine in organic combination. With the increased range of dosage afforded by the two new products, it will now be even more serviceable than before. "Tabloid" Thyroid Gland gr. $\frac{1}{10}$ and "Tabloid" Thyroid Gland gr. $\frac{1}{4}$ are both issued in bottles of 100 products.

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A LECTURE

ON

ABDOMINAL INJURIES.*

By V. ZACHARY COPE, B.A., M.D., M.S.Lond., F.R.C.S.Eng.,

Surgeon to Out-patients, St. Mary's Hospital, W.; Surgeon to the Bolingbroke Hospital.

GENTLEMEN,—There is no need for me to apologise for taking abdominal injuries as the title of my lecture to-day for the subject is one of constant and ever-increasing importance; this is especially true in the great cities, where traffic, more particularly motor traffic, has reached enormous dimensions, and brought increased risk of bodily harm to so many inhabitants who have to frequent busy thoroughfares. An additional reason for taking this subject might be found in the rather unsatisfactory way in which some of the modern text-books deal with it. In the short time at our disposal we can do no more than illustrate some of the main features in diagnosis and the chief indications in treatment. From the point of view of susceptibility of its contents to injury the abdomen can be divided into three parts: an upper zone lying under cover of the ribs, a lower portion protected by the bony ring of the true pelvis, and a median zone stretching from ribs to pelvis, the parts lying under which are the most exposed to external violence. Between the ribs and the bony pelvis stretch several muscle-sheets, which serve as a strong shield against external violence. Laterally are three layers of thick muscle (external oblique, internal oblique, and tranversalis), the fibres of which course in different directions, thereby giving additional strength to the abdominal wall. In the central portion of the abdomen are situated the strong recti muscles, separated by the linea alba and each divided into several portions by the tendinous intersections, so that contraction and rigidity of any one part may

* Delivered at the Polyclinic.

be economically brought about. Far back in the loin the thick erector spinæ bridges the gap between the ribs and iliac crest.

Injury of the pelvic contents is almost always associated with fracture of the bony pelvis, the discussion of which would take us too far afield; we shall confine overselves to the parts more exposed to injury, *i. e.* the upper and middle portions of the abdomen. The abdominal viscera are either solid or hollow. The solid organs (liver, spleen, pancreas, and kidneys), are situated above the level of the umbilicus; the hollow viscera (stomach, intestines, biliary and urinary channels), both above and below that landmark. Since the crushing of the solid viscera is fraught with more immediate peril, it is of advantage that they are partially protected by the costal framework; the hollow viscera hardly need protection so much, for they are comparatively mobile and elastic, and can frequently evade violence by slipping aside out of harm's way. This evasion of impending injury is possible for intestine supplied with mesentery, but if the strain occurs near a fixed portion of the gut, *e. g.* near the duodeno-jejunal junction, severe injury may follow. Again, though the mobile intestine may escape, yet the attached mesentery may bear the strain at its fixation-line to the posterior abdominal wall. This fact was illustrated in a very recent case within my knowledge in which the intestine was uninjured, but the mesentery was completely torn away at its roots. The large blood-vessels—aorta and vena cava with their branches and tributaries—lie far back in the abdomen, and are seldom exposed to injury without other viscera undergoing severe trauma. The mesenteric vessels constitute a partial exception to this statement.

Abdominal injuries may be due to penetrating or non-penetrating violence. Under the first heading come stab- or bullet-wounds, and some cases of blunt violence in which the abdominal wall gives way under the excessive tension produced. Non-penetrating or subcutaneous injuries can be produced in several ways. Most commonly they are the result of severe crushes, such as would follow from a heavy vehicle running over the abdomen. In the upper zone the lower ribs are liable to be fractured, and to be driven on to the subjacent viscera, thereby inflicting very severe injury. Another type of violence is the sharp blow due to a kick or punch, or the impinging of any circumscribed hard body against the abdominal wall. For example, a girl fell off her bicycle, of which the handlebar struck her in the upper abdomen; no bruise was visible externally, but she was found to have a ruptured intestine; in this case, as often occurs, though there was no external mark of violence, yet there was ecchymosis of the deeper parts of the abdominal wall seen at the operation. There has been some discussion as to whether the rupture of intestine following a sharp blow is due to the gut-wall giving way at the place of impact, or bursting at another portion of its circumference. Most likely it gives way where the injuring force impinges on it, for I have seen well-marked ecchymosis in the intestinal wall due to external violence, and it would need but a little extra force to have caused a complete rupture. If at the time of injury any of the hollow viscera are distended with fluid or gas, it is quite conceivable that they would give way at the weakest part of their wall, even away from the immediate region of the applied violence.

Sudden jolts can cause serious intra-abdominal injury. A fall from a height on to the feet, or flat upon the abdomen, or a sudden trip leading to a violent fall forward, may cause such strain upon the attachments of partly mobile organs that a tear occurs. I have known the renal vessels torn in this manner.

When violence is applied against a muscular part of the abdominal parietes the kind of injury produced depends to a certain extent upon the preparedness of the patient for the blow, and the consequent rigidity or flaccidity of the muscular layers. If the muscle is taken unawares more serious intra-abdominal mischief is to be expected, whereas if the muscle is rigid it may mitigate or prevent the injury of the deeper-lying viscera. The more the abdominal wall is injured, the less are the subjacent parts. Injuries of the abdominal wall comprise:

(*a*) Simple contusions.

(*b*) Incision or laceration of the superficial parts.

(*c*) Incision or laceration of the whole thickness of the wall—the so-called penetrating injury.

In addition to these it must be remembered that

(*d*) The deeper parts of the abdominal wall may be ruptured without the skin being broken, and the viscera may lie just under the skin.

This constitutes a traumatic ventral hernia.

Simple contusions of the abdominal wall are usually accompanied by tenderness and rigidity over the bruised area, but the rigidity as a rule passes off in from twelve to twenty-four hours if no other lesion is present. The persistence of rigidity betokens something more serious. In connection with small wounds of the abdominal wall made with sharp instruments, *e.g.* a knife, it must be remembered that an apparently superficial wound is often in reality a penetrating one. The utmost care is, therefore, necessary in examining such injuries, and in case of doubt an anæsthetic must be given, the wound enlarged and thoroughly explored. It is not sufficient to probe the external wound; the muscles contract, and may prevent the probe going through a slit made by the injuring weapon. This especially applies to stab-wounds. I cannot do better than illustrate this by recounting a case recently under my care. A man was stabbed in the lower abdomen during a brawl, and when admitted to hospital was sleeping off his drunken stupor. There was a small half-inch cut, just above the pubes, which was thought by the house-surgeon (a very capable man) to be quite superficial; the wound was carefully searched with a probe, which did not go to any depth. His pulse was 100 to the minute and there was no marked rigidity or tenderness of the abdominal muscles, so it was not thought necessary to inform me. For eight hours his condition remained the same; then he complained of acute abdominal pain, and his pulse rose to 120, so I was summoned. I found marked signs of intra-abdominal hæmorrhage and peritonitis. Laparotomy revealed a ruptured intestine and the peritoneal cavity full of blood. I sewed up the intestine, but could do no more on account of the patient's perilous condition. He did not survive long, and at the autopsy we found a rupture of the iliac vein. The knife had gone through the posterior parietal peritoneum some little distance from the vein, and the valvular opening had apparently prevented any bleeding into the peritoneal cavity at first. This case enforces what I think should be a general rule—that all stab-wounds of the abdomen should be opened up and explored under an anæsthetic. (The same does not necessarily hold good with regard to bullet- and shot-wounds, which I do not propose to discuss.) When the laceration or wound of the abdominal wall is considerable, portions of intestine may prolapse. This is not always associated with much shock. Three years ago a young man was admitted to St. Mary's Hospital with the history that the shaft of a cart had struck him in the right iliac region. When admitted there was a valvular wound with external opening near the right anterior superior spine, and the cæcum was prolapsed through the wound. Yet the man's general condition was very good. Under an anæsthetic the parts were cleaned, the cæcum replaced, the abdominal wound sewn up in layers, and drainage provided. He made an uneventful recovery.

The existence of *traumatic ventral hernia* is doubted by some, but it can readily be understood that if the fibro-muscular and deeper portions of the abdominal wall are ruptured the subjacent viscera may lie merely covered by skin. A case of this kind came under my observation some time ago. A man of fifty-seven years was crushed between the buffers of two goods-wagons. When admitted there were abrasions and contusion of the front and right side of the abdominal wall. Pulse 80. No other injury detected. For two or three days after admission he occasionally vomited, and blood was noted in the vomit. A more careful examination was then made, and showed that the abdominal muscle had been ruptured subcutaneously, and gut was felt immediately under the skin. This was confirmed at the operation which was undertaken. The ruptured muscles were sutured after the small intestine had been replaced. Such a case may be rare, but should establish the occurrence of traumatic ventral hernia.

Now let us consider those cases in which the abdominal wall does not present any marked sign of injury, but the subjacent viscera bear the brunt of the violence. The main symptoms which then follow are those of shock, internal hæmorrhage, and peritonitis.

Shock may be very severe, but unless there is some serious lesion, usually soon passes off. If the state of shock lasts longer and seems out of proportion to the evidence of intra-abdominal injury, careful examination will usually reveal another cause for such shock. Serious lesions of the chest must specially be sought.

Four months ago I was summoned to see a boy who had been injured by a motor. He was in a

condition of profound shock, and presented all the signs of internal hæmorrhage. There was tenderness of the abdomen and evidence of free fluid, but not quite enough to account for his pallor and collapse. Examination of his chest revealed a left-sided pneumothorax which speedily became a hæmo-pneumothorax. No operation was, therefore, undertaken, and the boy made a good recovery.

Injuries of the upper abdomen cause more serious shock symptoms than those of the lower portion. Renal contusions may cause alarming symptoms. I was once sent for urgently to see a man who had struck his loin against a large girder. He was said to be in a condition of profound collapse. By the time I arrived, however, the shock had partially passed off, and only hæmaturia remained to tell that the kidney had been badly bruised.

If shock does not pass off within six or twelve hours hæmorrhage or peritonitis is almost certainly present. Rupture of solid organs leads to the former, injury to hollow viscera to the latter.

Owing to the anatomical disposition of parts hæmorrhage usually follows lesions of the upper zone, in which lie the liver, spleen, and kidneys. The two former are most commonly injured. When the rupture is very severe the signs of shock and hæmorrhage commingle, and the patient dies soon after the injury. If the rupture is less serious the evidences of bleeding will be increasing restlessness and pain, progressive pallor of the lips and finger-nails, rising pulse-rate, and movable dulness in the flanks. Occasionally, as in one case which came under my observation, the signs of bleeding may cease for a day or two, and then become alarmingly evident after some exertion, *e.g.* straining on the bed-pan. It has been pointed out that the pulse-rate is not always a reliable guide in abdominal hæmorrhage. It may continue fairly slow till the abdomen is full of blood and then suddenly bound up to a rapid rate.

Bleeding is sometimes due to rupture of the great abdominal blood-vessels, but with the exception of the mesenteric vessels these lie too far back in the abdomen to be injured frequently. Rupture or contusion of the kidney may manifest itself in three ways—hæmaturia, intra-abdominal hæmorrhage, or retro-peritoneal hæmatoma. In most cases the lesion is slight and stops spontaneously, but occasionally the injury is so severe as to necessitate nephrectomy.

The hollow viscera may be either contused or ruptured; the stomach, intestines and bladder are most commonly injured, the gall-bladder and ureter rarely. Sometimes the injury is only sufficient to bruise the walls of the viscus. If the stomach is thus contused, the result is vomiting and, maybe, hæmatemesis; if the colon is bruised, bleeding from the rectum, and sometimes diarrhœa due to traumatic colitis, may follow. Bruising of the bladder causes temporary hæmaturia.

In considering the symptoms which result from a complete rupture of a part of the alimentary tract, the contents of the canal need to be recalled. The gut contains fluid, gas and microbes; the stomach and colon in addition may contain solid material. The stomach should contain few bacteria, owing to the presence of hydrochloric acid; the microbic content of the intestines increases from above downwards, and attains its maximum in the colon and rectum. If the stomach is ruptured on its anterior surface, there is an immediate escape of much free gas and fluid into the peritoneal cavity; general abdominal rigidity supervenes, free fluid is soon detected in the flanks, and the pelvic peritoneum becomes tender. The nearness of the liver to the escaping gas enables the obliteration of liver-dulness to be of some value in diagnosis. There are two signs which may be obtainable as the result of the escape of a large quantity of gas from the viscus. The first is a markedly tympanitic note over a circumscribed area in the region of the stomach due to the localisation of the escaped gas; the second is what, for want of a better expression, I should call an emphysematous gurgle. The latter is due to the mingling of gas and fluid between the intestines. It is detected in the left loin, and can be felt better than described. It indicates severe rupture, and may occur very soon after the accident. An epigastric injury followed by marked signs of peritonitis without indications of intra-peritoneal hæmorrhage usually means ruptured stomach.

Rupture of the intestine is a condition fraught with danger of almost certain death if not diagnosed early, and yet the signs and symptoms are often equivocal for some hours after the accident. Let me explain why this is so. When intestine is

injured its movements stop, owing to a reflex or direct paresis of its walls. If the rent is small, the edges of the mucous membrane pout and fill up the small gap; a little escape of intestinal contents occurs, and sets up a local plastic peritonitis with deposit of lymph, which leads to the agglutination of neighbouring coils of intestine. The patient takes nothing by the mouth, and the intestines remain at rest. A lull in the symptoms gives a false security. After a few hours, when it is thought that the condition is merely an abdominal contusion with no serious lesion, food is given by the mouth, the intestines are excited to peristaltic contractions, and the opening in the gut is unsealed. A spreading peritonitis then reveals itself. Another occasional reason why such injuries are not diagnosed early is that they may be associated with some other injury for which the patient is primarily admitted to hospital, or which attracts more attention. One of my cases was admitted with a Pott's fracture, and it was many hours later that the abdominal condition caused marked symptoms. What, then, are the main guides in the diagnosis of ruptured intestine? The symptoms readily follow from the pathological conditions. The initial shock of the injury is usually recovered from, but local pain, tenderness and rigidity remain. Vomiting (that almost constant sign of incipient peritonitis) occurs and becomes more frequent. The pulse and temperature rise, but, as a rule, rather later than one cares to leave the diagnosis in doubt. Nevertheless, the counting of the pulse at half-hourly intervals is necessary and useful. There are two other symptoms of importance, both depending on the fact that gravity directs the inflammatory fluid and escaped intestinal contents into the pelvis. The first is the tenderness of the pelvic peritoneum as felt *per rectum*. The second is only of value in upper abdominal injuries, and consists in the shifting of the site of pain and rigidity to the hypogastrium. The site of injury still remains tender and rigid, but not to the same extent as the lower abdomen. The obliteration of liver-dulness is usually of late onset, but may be of assistance in some cases. Diagnosis should usually be made before it is detected.

The prognosis of ruptured intestine without operation is hopeless, and even with operation the statistics show a mortality of 80 per cent. During the last year I have been fortunate in treating two cases, both of which made a good recovery. I will give you brief notes of them, for they illustrate the foregoing remarks.

H. O—, a young woman, æt. 20 years, was riding a bicycle at 9.30 p.m. on July 16th, 1912, when a motor knocked her off her cycle; her ankle was injured, but she returned home. About one and a half hours later she experienced pain in the abdomen, which had been struck by the handlebar. At 1.30 a.m. on the 17th vomiting occurred, and she was brought up to hospital on account of the pain. On admission attention was directed more to the ankle than to the abdominal condition, for the signs of the latter were not definite. Pain was felt in the left hypochondrium, but no vomiting took place. As the day went on abdominal pain became worse, but the pulse went down from 104 to 80. At five p.m. on the 17th I was called to see her; I found the lower abdomen rigid and tender, and not moving with respiration. There was pain at the site of injury under the left upper rectus. *Per rectum* the pelvic peritoneum was tender to the touch, the pulse was 80 to the minute, and quite good. No trouble with micturition; nausea, but no vomiting; temperature 102° F. I operated twenty hours after the accident by a supra-pubic median incision. A great amount of greenish alkaline fluid found in the pelvis. There was a little free gas. The pelvic viscera were intact. An epigastric incision was then made, and lymph found glueing some jejunal coils together. A perforation the size of a threepenny-bit was discovered on the anti-mesenteric border of the gut. The mucous membrane pouted into the opening, from which greenish contents were escaping. The gut was clamped on each side of the perforation, which was sewn up carefully after trimming the edges of the mucous membrane. The epigastric incision was sewn up without drainage, but a tube was put into the pelvis. After the operation the patient was propped up, and given the treatment now customary in cases of peritonitis. Four days later her pulse, respiration and temperature were normal, and an uninterrupted recovery followed. The epigastric incision healed by first intention. Four months later the patient came back to hospital with an attack of acute appendicitis. I performed appendicectomy, and took the opportunity of examining

the site of former injury, which had healed perfectly, and showed no signs of stenosis.

The second patient, T. H—, was a man, æt. 35 years, who was struck by a taxi-cab in such a way that his abdomen was forced violently against the edge of a board on the barrow which he was pushing. He was admitted about midnight suffering from shock and abdominal pain. Next morning the pulse had become regular and good, but pain and rigidity persisted. During the day the pulse-rate and temperature went up, and vomiting occurred several times. That evening hypogastric tenderness and rigidity were marked, rectal examination was painful, and percussion over the liver gave a resonant note. Operation, performed twenty-two hours after the accident, showed conditions worse than in the preceding case. The jejunum was ruptured, and there had been free escape of fæculent material. The gut was sewn up, the epigastric incision closed, and the pelvis drained. For several days the patient's condition gave anxiety, but, thanks largely to good nursing and the assiduous care of my house-surgeon (Mr. Martyn), a good recovery was ultimately brought about.

In the treatment of these cases I wish to emphasise the fact that I did not wash out the abdomen with saline solution, although the peritoneum was badly soiled with intestinal contents. To do so is, I believe, but to spread infection.

Ruptured bladder usually occurs in connection with a fractured pelvis, but may result from a blow on the hypogastrium when the viscus is distended. The symptoms vary according as the rent is intra- or extra-peritoneal. If intra-peritoneal, pelvic peritonitis ensues, but it must be recollected that sterile urine does not at first cause very acute inflammation of the peritoneum. There may be hæmaturia. If there is any doubt as to rupture a measured quantity of sterile saline should be injected into the bladder and note taken whether it all returns. Extra-peritoneal rupture leads to extravasation of urine and cellulitis in the supra-pubic and perinæal regions.

The question of treatment of intra-abdominal injuries is too big for us to consider fully in this lecture: it may be summarised thus. Tears of the liver are sutured or plugged. Ruptures of the spleen may possibly be sutured, but splenectomy is usually the best course. Renal injuries, if slight, are usually treated expectantly, but if severe may require a nephrectomy. Tears in hollow visceral must be sutured, but in the case of intestine if the rupture is large or the mesentery seriously torn, excision of the affected portion with end-to-end suture may be required.

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TWO CLINICAL LECTURES
ON
CALCULUS IN THE UPPER URINARY TRACT.*

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I. Renal Calculus.

Gentlemen,—As this is a clinical lecture I propose to deal with the subject of renal calculus purely from the clinical point of view, and shall not therefore go into questions of pathology, composition of stones, and so forth. So we will first discuss some of the symptoms and signs of renal calculus.

The first is pain, and I put it first for two reasons: (1) because pain is the commonest symptom of renal calculus; (2) because it is the most important symptom to the patient, and it is nearly always the one which makes him seek relief, for the great majority of the subjects of this condition come complaining of pain in the side. The character of the pain caused by renal calculus varies immensely, and I shall try to give you some idea of the variations in type and degree. In the first place the pain varies greatly according to the age of the patient. I mention this particulariy, because the pain of renal calculus in children is usually very slight or altogether absent, whereas in adults the pain may be very severe. I have only once operated on a young child for calculus in the kidney, a boy, æt. 9 years, whom I had under my care at the hospital in Great Ormond Street. He had a stone as big as the end of my thumb in the pelvis of his right kidney. But the interesting point is that that boy was admitted to hospital, not on account of renal calculus at all, but because he had a vesical calculus. It was in the early days of X-ray work at the hospital, and I had him skiagraphed, although he was known to have a stone in his bladder. The report to my surprise stated that he had also a stone in his kidney, and it was well shown on the plate. The boy's mother was questioned, and she said he had never complained of pain in his side, and there were no other signs of renal calculus present. So I dealt with the stone in his bladder, and asked his mother to

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watch him. At the end of a month she brought him up again, and said he had never complained, and I had him X-rayed again. Remember these were the days when one was sceptical about some of the results of X-ray examination. Again, there was no doubt that what the plate showed was a stone in the kidney. So I operated, and removed this large stone which I show you. I mention that case to impress upon you that stone in the kidney in children is frequently unaccompanied by pain. Another interesting point about cases of stone in children is that, with very rare exceptions, they are situated in one of two places—either in the bladder or in the urethra. In the male child the commonest clinical type of stone is an impacted calculus in the urethra. Probably one or two of these appear at the Front Surgery every month. Though I have frequently inquired into the history of these cases, I have never heard of an attack of renal colic or renal pain suggesting that the boy had anything the matter with him before, although it was clear that the calculus came down from the kidney.

Next, the pain varies with the *position* of the stone. A stone may be in the cortex of the kidney, or in its pelvis. Dealing first with those in the kidney cortex, the pain in that event is usually comparatively slight, but it is more constant than in the case of stone in the pelvis of the kidney: there are not the violent attacks of renal colic which I shall describe presently. I may mention at once a case which illustrates this. It was that of a man whom I saw some years ago; he was sent to me by an old Guy's man to the Out-Patient Department on account of pain in his back. He had been treated by various people, and had been to several other hospitals. The pain was more marked on one side than on the other—I forget which side. He had previously been treated with liniments and blisters, probably without his case having been very carefully gone into. I suspected that there was some underlying cause for his complaint, and I remember speaking of stone in the cortex of the kidney as one of the causes of chronic pain in the back. X rays showed a stone apparently in the outer border of the kidney, so I took him into hospital and removed it. Here it is. It is a small, round, dark oxalate of lime calculus, and was embedded in the outer part of the cortex of the kidney. It had caused constant aching pain for several years, and, so far as we know, it was not associated with any other symptom. I will tell you of another case which illustrates this kind of stone. The patient was under the care of Dr. Pitt some years ago, when I corresponded to him, and was a woman who had been in the hospital some time on account of pain in the back. She had been skiagraphed and her urine examined with a negative result, and she had been treated in various ways, but nothing that was done made any difference to the pain in the back. Yet it seemed a genuine case, not functional. Dr. Pitt asked me to discuss the case with him, and there was no doubt her pain was in the renal region; but it was not associated with tenderness or any urinary changes; moreover, no stone showed on X-ray examination. However, her symptoms were so constant, and had resisted so many forms of treatment, that it was decided to make an exploratory incision to see whether there was any relievable cause to be found. The kidney was exposed but I could not feel anything abnormal, so I incised the convex border of the kidney, and palpated all over from the inside. I was on the point of giving it up when I felt just a small point at which the consistence was different from the rest. It proved to be the edge of a very narrow stone, lying from before backwards in the cortex of the kidney. The removal of the stone entirely cured the pain.

Next, let us consider cases where the stone is situated, not in the cortex of the kidney, but in its pelvis. The first point I want you to note about these is, that the symptoms produced by a stone in the pelvis of the kidney vary according to the size of the stone. And that is a very useful guide, because you will find it applies also in biliary calculi. Supposing the stones are very small—and as a rule they are multiple when very small—the patient may have a sharp attack of pain, lasting five or ten minutes, starting in the loin and possibly running down to the groin, and then it may be all over; and she may take no further notice until seized with another attack. I had, in Lydia ward some years ago, an interesting case which illustrates this point very well, I shall have occasion to refer to it again later from another point of view. She went originally to the out-patients of the late Dr. Bryant for these attacks of sudden pain, but nothing could be found to

account for them, and Dr. Bryant was not sure of the diagnosis. The pain was on the right side. However, he suspected the possibility of either biliary calculus or renal calculus, and advised her to examine her urine and fæces carefully after an attack. She did so, and found that after the pain she passed a little stone, such as I show you. These stones vary from $\frac{1}{32}$ to $\frac{3}{16}$ of an inch in diameter. After she had had one or two more attacks I admitted her into Lydia ward, and operated upon her. She had more than 100 of these stones in the pelvis of the right kidney. Her symptoms were sudden violent attacks of pain which made her feel bad, but were quickly over, and she had no hæmaturia or other symptom. The explanation is that these stones, being very small, passed rapidly down the ureter.

If the stone is bigger and gets into the ureter it takes more time to pass down; and the ureter contracts more violently, so causing a severe attack of typical renal colic. About eighteen months ago, when I was doing duty in Clinical, I saw such a case, which had just been taken into John. The history was that he had been suddenly seized with a most violent pain in his left side, he had been sick, and he was then lying doubled up with pain, although he had had morphia. He had a very feeble pulse and cold clammy skin, and was writhing in agony. This lasted for some time, in spite of a further dose of morphia. After the pain subsided he passed a good deal of blood in his urine.

If the stone is a little larger still, it may, instead of passing all the way down, become impacted in the ureter. In these cases the pain usually passes off gradually, not suddenly. It may be quite absent for a day or two, and then recur from time to time, as the ureter, as it were, gathers strength to have another push at it. So the pain in this case is of the recurring type. Not very long ago I had, in Lydia ward, a woman who had such a condition. She had a tumour in her loin, which was due to hydronephrosis, associated with calculus in the ureter, and I show you a skiagram of the case. She had had pain for several months, beginning with acute attacks, and associated with exacerbations. X-ray examination showed an oval stone lying in her ureter, at about the level of the brim of the pelvis. I removed the stone, of which I show you a photograph. It gives you an idea of the size of stone which may be impacted in the ureter. When there is a calculus a little larger still it cannot pass into the ureter, and so has to remain in the pelvis of the kidney; and then there is again a different type of pain. There is pain which lasts a few hours at a time, and is brought on by exercise, or even by movement: riding in a 'bus, or even in a train, will also bring the pain on. The stone may be irregular in shape. I show you a stone which was removed from a man who had no renal colic, but had had frequent attacks of renal pain. One corner of such a stone may get caught in the orifice of the ureter, and give rise to an attack of pain, lasting any time from a few minutes to a few hours, till the stone falls back into the pelvis of the kidney again. These attacks of pain are not like the constant ache of a stone in the cortex. They are more severe attacks, but not so severe as typical renal colic. There is nearly always hæmaturia after the pain.

In 1910 a patient was admitted under my care on account of pain in the back, which he had had for some time, and which became worse on walking. Probably other things brought it on as well. It spread round from the left side, and had been associated with hæmaturia on four occasions, the first time eight weeks previously. He had had three further attacks at intervals, the last a week before admission. X-ray examination showed a stone in the pelvis of the kidney, and I show you the stone which I removed.

If the stone is larger still it may cause no pain at all, because it fits into the pelvis of the kidney so tightly. So that pain may be due, first of all, to the contraction of the ureter in renal colic; secondly, to the gripping of the hard surface of the stone by the kidney pelvis, and the congestion which is set up by the presence of a foreign body. And finally a very large stone may cause no pain at all. Here are two enormous stones that I removed from the pelvis of the kidney in a case where there was no pain at all. His history was that thirty years previously he had an attack of renal colic, but had not felt anything the matter with his loin since that time. As a matter of fact, he was admitted on account of multiple joint disease associated with some pus in his urine, which was found to be due to these stones.

Now we come to the other symptoms. I put hæmaturia next, because it is the next commonest.

The hæmaturia of renal calculus is very frequent, and is of two types. First, there is the hæmaturia associated with renal colic; it is always present where the colic is severe. Then there is the type of hæmaturia brought on by exercise, or jolting, when the stone is lying in the pelvis, or fixed in the cortex. In cases of renal calculus you will nearly always find that the hæmaturia is associated with pain, and I want you to make a special note about that, because it is very useful in diagnosis. The next most frequent cause of hæmaturia is tuberculosis of the kidney, but as a rule this is not associated with pain. So in most cases you will be right if you say hæmaturia + pain means renal calculus; hæmaturia – pain means tuberculous kidney.

Now we come to pyuria, which, if looked for, is found fairly commonly in cases of renal calculus. Of course, it is due to the fact that stone in the kidney sets up some irritation, being a foreign body, and that irritation causes pus-formation. In some cases the amount of pus is very large, owing to suppuration of the kidney as a whole.

The following case is an example of this condition. The patient, a woman, was very emaciated, looked very ill indeed, and had had a high temperature for some weeks. On examination one could feel a large tumour in the right loin, and there was abundant pus in the urine. I explored the tumour, and found it to consist of a huge pyonephrosis, seven or eight large calculi lying in a loculated bag of pus. The kidney was removed, and the patient did very well.

Next, I want to refer to calculous anuria. If one kidney is completely destroyed by renal calculi, the other kidney may be put out of action by a calculus which has become impacted in the ureter, with resulting anuria. Though such an event is rare, it is important to remember the possibility. A number of cases of anuria have been recognised as due to calculus, and have been operated upon successfully. If a patient gets anuria, therefore, remember that there is not much time to lose; and to recognise calculus as a possible cause may result in the saving of the patient's life.

In cases of suspected calculus palpation is very important, and in the first place you may find that the kidney is tender as a whole. Or if you do not find general tenderness you may, especially in the cases in which there is a stone in the cortex of the kidney, find deep tenderness at one spot. If you systematically go over the loin exerting fair pressure with your thumb, you may come to one spot where the pressure causes the patient to jump. Palpation may also reveal the fact that the kidney is enlarged, and that enlargement may be due to the presence of pyonephrosis. Or it may actually be palpable from the size of the stones. The gentleman I have spoken of who had these enormous stones is a case in point. He was very thin when he came to me for examination, and I could feel the stones very well through his abdominal wall. Very rarely in the case of multiple calculi in the kidney you can actually feel the stones grating against each other. I have only felt that once, and it was in the woman I told you about, who had these tiny stones; on pressing firmly while she breathed deeply, one could feel the stones grating against each other quite plainly.

Now, as to X-ray examination. To what extent is that method of examination reliable in the case of stone in the kidney? I think you can say this now, that if the result of X-ray examination is positive, *i. e.* if a stone shows on the plate, that is reliable evidence. A few years ago the position was not quite so clear. On the other hand, you should not necessarily accept negative evidence, *i. e.* if X-ray experts say they see no evidence of stone, it does not prove that there is no stone present. With regard to the circumstances which may produce this negative result, the first is, that the composition of the stone may be such as to account for the lack of shadow; some calculi are much less opaque to the rays than are others, the most opaque being oxalate of lime, and the least opaque urate calculus. Again, the stone may be so small that it does not cast a recognisable shadow; the narrow stone which I showed you is a case in point. Or the patient may be so stout that it is very difficult to pick out a definite shadow; and the same may be true if there is a pyonephrosis.

Finally, I have had one case in which the failure was due to the stone being in a horse-shoe kidney.

The patient was in Job ward in 1910. He had attacks of pain on the left side, but also to a slight extent on the right; and had twice been X-rayed on the left side, but with a negative result. He

had had several attacks of hæmaturia, and had another in the ward. Another X-ray examination was therefore made, and there was the stone showing on the plate quite plainly. At the operation I found that he had a horse-shoe kidney, and that the stone easily rolled away to the other side. That accounted for his having had pain on both sides, and for the fact that the stone was missed on two X-ray examinations. If both sides had been X-rayed the stone would probably have been found at the first attempt.

Treatment must be dealt with very briefly. If there is but one stone and it is comparatively small, you may tell the patient that he need not have it removed at once, as the effect of solvents can be tried. There are a number of solvents on the market, but I don't think they are of much value. The only important solvent is water. If the patient is a poor man, get him to drink large quantities of distilled water. If he can afford it and is anxious to go abroad, send him to Contrexèville. There is no doubt that numbers of stones are passed after taking Contrexèville water. I have known several cases in which patients were advised to try a "cure," and have passed their stones. But I should only consider that course if the stone be small and single. If the stone is large, or if there are several, you should advise operation, because they will sooner or later cause destruction of the kidney; moreover, the operation is, nowadays, very successful and practically devoid of risk.

March 24th, 1913.

Sudden Whitening of the Hair.—In the case reported by Loeb a man of 51 became paralysed on the left side in consequence of a cerebral hæmorrhage and was unable to speak for a week. By the end of this time the moustache, eyebrow and hair on this side had turned entirely white, while on the other side the normal colour had been retained. Berger has reported two similar cases of the hair turning white on the side of the paralysis. Loeb regards this as sustaining the assumption that nervous influences alone are able to turn the hair white suddenly.—*Journ. A.M.A.*, vol. lx, No. 8.

A LECTURE

ON

THE EARLY RECOGNITION OF PULMONARY TUBERCULOSIS.*

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GENTLEMEN,—The fight against tuberculosis is at present very much to the fore, in both professional and lay circles. A serious effort is being made to work the Sanatorium Benefit under the National Insurance scheme, and the large chest hospitals in London are co-operating with the local authorities in order to render those institutions which have stood the test of time of assistance in combatting the disease, rather than that new and untried dispensaries entirely under the control of the municipal authorities should absorb a revenue which might be better expended. Further, the Royal College of Physicians of London is urging the teaching schools to establish a special department under their management for the study and treatment of tuberculosis, so that students may have early and frequent opportunities of seeing patients who are suffering from pulmonary tuberculosis. The principal chest hospitals, also, are organising courses of lectures and demonstrations for the benefit of the post-graduates.

It would be needless for me to urge that, for the purposes of treatment, early diagnosis is of the utmost importance; although we must not be led to the opposite extreme and say that cases, for instance, in the cavity stage, are perfectly hopeless. They are by no means so, provided that auto-inoculation has ceased. Some of those cavity cases do very well. And I think that perhaps the expression "early stage" may be misleading. There are two or three expressions which, in agreement with some other physicians, I should like to see disappear. One of them is "phthisis," for which we want to substitute the more cumbrous but more exact term, "pulmonary tuberculosis"; and the other word is "cure," substituting for that, "arrest." I should also like to see the term

* Delivered at the Polyclinic.

"early case" done away with, because it is likely to lead to misunderstanding in prognosis. It is quite possible to have two persons standing by each other, one with only a few signs at the apex, and the other with a cavity which is undergoing arrest. We know what has happened to the man with the cavity, but we do not know what will happen to a man who has a few *râles* at the apex. His case may be extremely acute, though early. So the term "early case," as used at present, may give a false impression of the condition present. I am not to-day speaking of treatment, but I want to draw your attention to some points in reference to early recognition of the disease.

Let me say at once that I do not at all recognise the term "pre-tuberculous stage." I do not think that any such stage has been proved to exist. We are told that the pre-tuberculous stage is characterised by certain symptoms: general malaise, headache, pains in the limbs, anæmia, digestive disturbances and so on. A great number of these cases react to subcutaneous injections of tuberculin, and if they are treated by tuberculin magnificent results follow! But how anybody with any experience of pulmonary tuberculosis can claim 100 per cent. of cures in any stage of this disease I do not know; such a claim, I maintain, is totally unscientific. I think physicians who diagnose cases as pre-tuberculous forget the dictum given so many years ago, "*Tota ars medici est in observationibus.*" The physician may think only of the lungs, and does not make a further examination. I feel sure that if many of these cases were more carefully examined, a large percentage of them would be found to have some ailment of the digestive tract. In a large number of them they would find pyorrhœa alveolaris, in others atonic dyspepsia, and in others chronic gastritis. If the patients were properly treated for these conditions, the supposed pre-tuberculous stage would, I believe, disappear. Therefore, in this lecture I shall not take into consideration any pre-tuberculous stage.

For the diagnosis of ordinary active pulmonary tuberculosis the means at our disposal may be divided into two classes. First, there are the ordinary means of diagnosis, by which I mean the history of the case, the symptoms, and the physical signs. Secondly, the auxiliary means, *i. e.* examinations of the sputum and the blood, and the use of tuberculin for diagnostic purposes.

First let us consider, then, the ordinary means of diagnosis. Here I would say that in the experience I have had of faulty diagnosis, I have found the sin of commission more frequent than the sin of omission. I see more cases which have been diagnosed as pulmonary tuberculosis when the disease is not present than cases of pulmonary tuberculosis being diagnosed as some other disease. In speaking of the ordinary means of diagnosis the first thing I would insist upon is, that we cannot diagnose the disease by any one symptom or any one sign; there must be a progression of symptoms and an assemblage of signs. That matter I shall refer to again.

The history of the illness will, of course, lend much aid, but there must be a progression of the symptoms complained of. The family history may or may not give us help. The absence of hereditary indications, needless to state, is of very little help. The death of one parent or of one brother or a sister from the disease is of but little significance, especially if some time has elapsed since that death took place. But the death of both parents from pulmonary tuberculosis, especially if one or more brothers and sisters have also been affected, is naturally more important, as tending to show that there is, apparently, a hereditary lack of immunity to tuberculosis. But when the parents are healthy and three or four brothers and sisters have been attacked, and especially if they are all living in the same house, one would naturally think of infection rather than of heredity. The theory of heredity is gradually losing ground, and not nearly so much stress is being laid upon it now as was the case some years ago. In getting the history of the patient, I think our questions should have reference to one or two specific diseases, and one of those certainly is pleurisy. It is now generally accepted that by far the most frequent cause of pleurisy is tubercle, and in a large number of cases of pulmonary tuberculosis a history of pleurisy is given. Therefore I lay much emphasis on the point as to whether the patient has or has not had pleurisy, although that event may have occurred some years back. And the importance of that is enhanced if the pleurisy was accompanied by effusion. The weakening of the general health by pneumonia,

typhoid fever, influenza, and so on, is sometimes found to immediately precede the commencement of the tubercle, probably by lowering the resistance to the *Bacillus tuberculosis*. But we come to this conclusion: that the chief practical point in connection with the history is that of pleurisy, possibly also the influence of the other diseases I have mentioned.

Now with regard to the present symptoms. Cough is the symptom which usually attracts attention first, but I need scarcely say that simply because a person has a cough, or has had cough for some time, this fact cannot lead to a diagnosis of pulmonary tuberculosis. There is nothing characteristic about the cough of early tuberculosis to distinguish it from the cough associated with other conditions. It is usually a hacking cough, and can generally be controlled by the patient. Also, it is apt to cause vomiting, especially after meals. But remember that from the cough alone we can come to no conclusion.

The next question is that of the expectoration. In the early stages there is nothing characteristic in the appearance of the expectoration. It is generally thin, sometimes frothy. In the later stages—which we are not discussing to-day—it is more characteristic. Even cough and expectoration together do not justify a diagnosis of pulmonary tuberculosis. Of course, we try to make a differential diagnosis by examining the throat more particularly.

The patient may complain also of loss of flesh. We must ascertain not only the amount of loss, but also its rapidity. I recently had a case which was sent to me at the Brompton Hospital with the diagnosis of tubercle, whereas it turned out to be a case of carcinoma of the rectum. It shows a neglect of the proper inquiries. Those three symptoms which I have mentioned—cough, expectoration, loss of flesh—may be due to other diseases besides tubercle. Even when anæmia is added to those, the conclusion that the person has tubercle may be fallacious. We find many cases which those symptoms are present, but where the patient is suffering from some other affection. But it must be admitted that the gradual progression of these symptoms in a patient increases one's suspicion that pulmonary tuberculosis is present. And in addition to these there may be general debility. Of course, if those five are present together, the significance is greater than if there is one alone.

We now come to consider a still more significant symptom, namely, hæmoptysis. When we say a patient has hæmoptysis, we must be quite sure that it is true hæmoptysis—that it is not spurious. I have seen several cases, generally in boys or girls of twelve to sixteen years of age, whose parents have been considerably alarmed because there is slight expectoration, and there has been some loss of flesh. The patient, especially in the case of a girl, is anæmic, and begins to spit up what the parents consider to be blood. But if you ask to see some of the matter brought up, you find it has a peculiar appearance; it is simply a thin, watery, pink fluid. The microscope does not reveal any cell elements at all, except some pavement epithelium. If you inquire more minutely about that, you find that the pillow is sometimes found stained with the fluid; it is simply blood-stained saliva. If the subject be a girl of sixteen to eighteen years of age, the occurrence sometimes synchronises with the menstrual period. Occasionally, if you examine the mouth carefully you find the gums are spongy, and very liable to bleed. In some of these cases the arterial pressure is found to be high. In short, these patients are simply spitting up saliva stained with blood which has oozed from the gums. It is scarcely noticed during the day, but during sleep this small degree of oozing accumulates, and is expelled first thing in the morning. These cases cause a good deal of suspicion, yet the subsequent course of the case shows the condition was not pulmonary tuberculosis. Sometimes a little streaky hæmoptysis is present. Some of these patients are often found to have varicose veins at the back of the pharynx, and on one occasion I actually saw blood oozing from one of these varicose veins, which was removed on a pledget of cotton-wool. People may have all the symptoms which I have mentioned, and yet not have tubercle. Therefore, you will see the need for being cautious in giving a diagnosis. But, if there be hæmoptysis of more than a drachm, especially if accompanied by the other symptoms I have named, the possibility of the presence of tuberculosis is certainly strong. Hæmoptysis above a drachm in quantity, even alone, I regard as the most significant of the early symptoms of tuberculosis. It is not absolutely

conclusive, but I think that in 95 per cent. of the cases tuberculosis is present. There are, of course, the remaining 5 per cent. to be dealt with.

With regard to pain in the chest, that is very variable. But if the chest pain is confined to the upper part, and especially if it be combined with tenderness, it is of some value, and particularly if associated with the foregoing symptoms.

Night-sweats often give cause for alarm, and patients are brought not infrequently because the mother states that the child sweats so much at night. She had been told that the child has consumption because of that symptom. But remember that night-sweats occur in all debilitating diseases. Some of the most profuse sweatings occur in connection with heart disease, especially mitral stenosis, and therefore they cannot be said to be necessarily a symptom of pulmonary tuberculosis.

Shortness of breath or dyspnœa is obviously, in the early stage of pulmonary tuberculosis, scarcely worth noting. So far the most important symptom I have mentioned is hæmoptysis.

Now we come to the most important of all, namely, temperature. There are, of course, numerous febrile diseases, but there is a peculiarity about the temperature in an early case of pulmonary tuberculosis which you can sometimes trace and which is of considerable value.

There is what I consider a significant chart, namely, a sudden rise of temperature followed by a flat curve; it is, I consider, more characteristic than any other type of chart. And if you watch it well, more particularly in hospital cases, where the temperature is recorded so regularly, you find that these rises occur after some unusual event. Perhaps the patient, after having been long in bed, gets up for a couple of hours, and then the temperature rises, to drop again when he returns to bed. The same thing happens following the excitement of visiting day. You can trace the auto-inoculations in this way, and they may be beneficial. In the early stage, with the patient lying in bed, he may not be able to control his auto-inoculations, and he has therefore the usual hectic type of temperature. That is a bad prognostic sign, while the other type I have mentioned is of favourable prognosis.

The value of the symptoms I have mentioned depends on the degree in which they are combined; it depends on having not one sign only present, but an assemblage of signs. I ask you not to be led away by one sign only. Let your examination be a most thorough one. For the examination of the chest the patient must be stripped to the waist. And examine every other organ in the body, especially the digestive tract, and do not forget to examine the mouth, teeth and gums for pyorrhœa alveolaris. It is wonderful how many patients complaining of headache, aching in the limbs, etc., are better when the teeth and gums have been attended to. Or there may be a chronic throat condition which requires attention.

And now with regard to the signs. One sign is of no use for diagnosis, but an assemblage of them is of great importance. One frequently sees cases in which pulmonary tuberculosis has been diagnosed, but when one puts them to a severe test and watches them over a period of a fortnight or three weeks, no pulmonary tuberculosis is found present. The cause of that mistake in diagnosis being made is forgetting the physiological departure from the usual signs of the chest. We are usually taught that the normal breath-sounds in the chest are vesicular, that is to say, they are the sounds conducted from the glottis and altered by the alveoli of the lungs, and so the character of the vesicular breathing is brought about. But remember that although vesicular breathing is normal, in certain parts of the chest we get other than vesicular breathing, even though the lungs are quite healthy. You must remember that the right bronchus is a continuation of the trachea, whilst the left bronchus is a branch, and comes off from it at an acute angle, with the result that the glottic sounds are conducted better down the right bronchus than down the left. In consequence there are often altered breath-sounds under the right clavicle. There may be simple harshness of breathing at that spot, or there may be only blowing sounds there. Occasionally there is typical bronchial breathing beneath the right clavicle, though no disease of the lungs is present. That is a danger in attempting to diagnose on one sign only. I have often asked students to examine a man, with the result that they say he has got a vomica or cavity. Where? I ask. At the upper part of the right lung, is the reply. Why do you think it is a cavity? Because there is bronchial breathing under the right clavicle. That mistake is made again and again. We must remember that altered breath-sounds under the right clavicle only

is no necessary indication of disease. We also find this type of breathing over the seventh cervical vertebra, yet there may be no other signs in the chest at all. The sound may extend to a quarter of an inch on either side. If it is found symmetrically on both sides you can place but little reliance upon it. If on one side only it is probably due to lung disease. Thus it is very important to remember those physiological variations from the ordinary breath-sounds.

Coming now to the systematic examination of the patient's chest, it is sometimes difficult to distinguish arrested disease from early disease. As regards the presence of enlarged glands, I consider that it is comparatively rare in adults with pulmonary tuberculosis. Perhaps the reason is that gland tubercle is generally caused by tubercle bacilli of the bovine variety, whereas in lung disease the bacilli are usually human in origin. The first thing we have to notice on looking at the chest is as to whether there is flattening. This is difficult to distinguish in the early stage. But there is often a deficiency of movement which can be detected. It is not easy to distinguish by simply looking at the patient, or even putting the hand on the front of the chest while you get the patient to breathe deeply. But if you stand behind the patient, and put your hand over the shoulders, the second finger below the clavicle and the index finger pressing down deeply above the clavicle, and then get the patient to breathe deeply, you can feel the apex of the lung distinctly hardening between the fingers, and you can appreciate the difference in expansion by that device.

Next to that, we generally test for tactile vocal fremitus. And there we must remember the differences in the ordinary physiological range. The tactile vocal fremitus is more marked on the right than on the left side. Therefore, if we find it more marked to the left it is an important fact.

With regard to percussion, the only hint I can give you is that the best results are undoubtedly obtained by very light percussion. The lighter the percussion the better your result. I have adopted the method of percussing almost entirely from the wrist or finger-joint, certainly not from the elbow. Our best percussors do it so lightly that practically only they themselves clearly appreciate the sounds elicited. One hears much about the significance of deficient resistance, but that is difficult to obtain. Some say you can feel the difference under your finger, others that you must percuss more heavily to obtain it. Percussion of apices has been described by some physicians, but the necessary skill in percussion is difficult to acquire.

With regard to breath-sounds, I have warned you against mistaking physiological differences for morbid ones. The earliest deviation from the normal in tubercle is, probably, prolongation of the expiration. Sometimes a rather harsh, wavy inspiration is heard, but that is only of value if it is heard at one apex only. If heard on both sides the cause is probably nervous. After that there may be what I would term broncho-vesicular breathing. The German name applied to it is non-committal, namely, "indeterminate." With regard to bronchophony, the voice-sounds may be increased in the early stages. But here, again, we must remember the need for an assemblage of signs for the purposes of diagnosis. Slight alteration of the percussion-note goes for very little, and the same may be said about altered breath-sounds, especially at the right apex. But if we find both these signs we are brought nearer our diagnosis. If, in addition, there are adventitious sounds, such as crepitant *râles*, the diagnosis approaches to certainty. If we find diminution of expansion, altered breath-sounds, *râles* after coughing, especially if there be also a progression of symptoms, we can give an almost certain diagnosis from the physical signs and history only.

But, especially in these days of notification, in order to make our diagnosis sure we have to adopt auxiliary means. Some of these can only be carried out in a laboratory. Others may be done by the practitioner himself. The first of these is the examination of the sputum for tubercle bacilli. It is accepted that if bacilli are present there is tuberculosis somewhere in the respiratory tract. But a negative result is of very little value. In one instance I examined nineteen specimens without finding tubercle bacilli at all, but on the twentieth trial I found them, and on the twenty-first attempt I missed them again. I could not examine again, because the case proved fatal. At the post-mortem miliary tubercle was found. I shall not now go into the method of examination, but simply say that if, with a progression of symptoms and an assemblage of signs we strongly suspect tubercle, and examination of the sputum has proved negative,

even on two or three occasions, it is advisable to collect all the sputum of several days and send it for examination by the antiformin method. Twenty per cent. antiformin solution is mixed with an equal quantity of sputum. That is heated and kept at a warm temperature for twenty-four hours, then put into a centrifuge machine and the deposit examined. In a goodly number of cases, where examination has previously proved negative, by taking ten days' sputum and mixing with antiformine the bacilli were found.

With regard to cytology—examining the blood for various kinds of cells—I need not dwell upon that this evening, as it is probably not of very much value.

I now come to a matter which has caused a good deal of discussion, namely the use of tuberculin for diagnostic purposes. There is first the ophthalmic reaction. Various tuberculins have been employed, but probably the best and most reliable is the old tuberculin of Koch, containing glycerine and phenol. For adults the old tuberculin should be diluted so that a 1 per cent. solution is obtained for the first test. For the second instillation 2 per cent. or 4 per cent. dilution may be employed. A positive reaction shows, in general, the presence of tuberculosis somewhere in the body, but it does not necessarily mean that the disease is in the lungs. It is one which I neither employ nor recommend, for even if positive you are none the more forward with regard to disease of the lungs.

Then we have the cutaneous or von Pirquet reaction. There Koch's old tuberculin, used undiluted, is employed. I like it done in this way: Take a sterilised needle and make, on the arm, two scratches. Put a drop of tuberculin above and between these scratches and draw the needle through again, crosswise. Thus you have two scratches through the tuberculin, and two control scratches, and if, after twenty-four hours, there is a reaction, as shown by a red swelling along the line of the scratches, that is positive reaction. It only shows that at some time or other during that person's life he or she has had tubercle; it does not follow that the disease from which he is now suffering is of tuberculous nature. A large number of people respond positively to the cutaneous reaction, and it is only of real value in young children, and in them its value is considerable.

Now I come to the real tuberculin test. That has given rise to much discussion. There are three effects of the subcutaneous use of tuberculin. First, the local reaction—that is to say, at the site of the injection; next, the focal reaction, as shown by the physical signs occurring in the lungs; and the general reaction as shown by the rise of temperature, and sometimes headache and other symptoms. I will say at once that I do not consider a mere rise of temperature after a single injection of tuberculin is of any value or significance as indicating that the person is suffering from tubercle of the lungs. Competent scientific observers all over the world are agreed upon that. And to say that a person should undergo a regular course of tuberculin because of one rise of temperature is quite unjustifiable. There have been numerous investigations of this kind, perhaps the most notable being those of Franz, who took a regiment of cavalry, four hundred in number, and injected them with tuberculin: 70 per cent. reacted by a rise of temperature. But would anyone be ready to say that those 70 per cent. should therefore be submitted to a course of tuberculin? But the matter is different when one comes to speak of the focal reaction. The patient's chest is examined in the ordinary way. There are no adventitious signs to be made out. A dose of tuberculin is given, and, in addition to the rise of temperature, there is a general reaction — headache, slight shivering, etc., and *râles* can be made out at the apex, where they were not heard previously. I think the focal reaction with the general reaction is of some value.

There is another way of employing tuberculin for diagnostic purposes which is of some value. But the patient must be kept in bed, and I prefer that he remain there during the whole course of the treatment. We begin with an injection of two tenths of a milligramme of old tuberculin. If there is no rise of temperature after that, we increase it to 1 mgrm. If still there is no rise, it it increased to 5 mgrm., and if still no rise, we give 10 mgrm. To be of value, as I say, the patient must be kept in bed, because if he is walking about we cannot say a rise of temperature is due to the tuberculin; it might be due to auto-inoculation. The dose is to be increased only if there has been no reaction from the previous one. If there is a reaction, the same dose may be

repeated. If the rise is due to the presence of tuberculosis, the same dosage should produce a more marked reaction than the first injection. Given in that way, tuberculin is of some value in diagnosis. Some people lay stress on the fact that there is a local reaction, that is, at the site of injection.

Personally, I very rarely use tuberculin for purposes of diagnosis. I only employ it as a last resort, because I think its indications are so fallacious. I prefer to use the test afforded by the opsonic index. An obvious disadvantage of that is that it can only be carried out by an expert. The practitioner procures the specimen of blood, but the expert does the test, and the expert must be one in whom one feels the greatest confidence. The specimen of blood is taken in the morning whilst the patient is in bed. Then he gets up, has his breakfast, and goes for a smart walk. After he returns another specimen of blood is taken, and those two specimens are compared. The normal may be taken as anything between 1·2 and — ·8. Between those points is the healthy swing of persons in health, as the result of exercise. If the index is above or below those figures, I consider it as diagnostic of pulmonary tuberculosis. Inducing auto-inoculation in that way is one of the earliest signs of the disease. I have had several cases in which the physical signs have been suspicious, and in which either there was no sputum available or it did not apparently contain tubercle bacilli and the opsonic index was positive, and after two or three months' watching of the case tubercle bacilli were found by the antiformin method. In one case a man obviously had gangrene of the lung. Was it a tuberculous cavity? Or, as he had had pneumonia, was it due to the pneumonia? We found no bacilli, but the opsonic index persisted in saying there was tubercle, *i. e.* taken in the way I have described, taking a specimen of blood first thing in the morning and then again massage (as the patient could not walk), it showed a distinct abnormal swing. The case proved fatal eventually. There were no naked-eye appearances of tuberculosis, but specimens of the pus from the gangrenous cavity showed numerous tubercle bacilli. After a long experience with the opsonic index test, I consider it one of the most valuable we have in the early stage of tubercle, when you are unable to get any sputum or unable to find the bacilli in it.

March 24th, 1913.

Botulism.—Schumache reports an episode of botulism attacking six persons. A man and wife were taken with colic in the night, but went to work as usual. They were soon compelled to return home on account of prostration and vertigo. The colic had not led up to diarrhœa or vomiting. Dysphagia next set in with hoarseness and dyspnœa. During the first day there also developed bilateral ptosis, which was the forerunner of numerous other eye symptoms—mydriasis, diplopia, paralysis of accommodation. There was persistent constipation with retention of urine. One physician diagnosed diphtheria in consequence of associated hoarseness and dyspnœa, and complete inability to swallow. These symptoms were, in fact, the threatening part of the disease. There was no constitutional reaction in the ordinary sense, as temperature and pulse were normal throughout. Pilocarpin gave some temporary relief. The man died on the sixth day with clear sensorium. Autopsy within twenty-four hours showed marked congestion of the internal organs, fluid state of blood, and œdema of lungs, which last had apparently caused death. Diphtheroid membrane was present in the throat. The mother of the patient died in a similar way, having presented paralysis of deglutition and speech disturbances. The patient's wife, already mentioned, recovered after presenting the same train of symptoms, but speech disturbances and slight paresis of the right hand remained. Three children of the family were ill, but recovered.

The malady was clearly one of wholesale poisoning, or infection. Rabies was first readily excluded, as was mushroom poisoning; poisoning by the atropine group and by hemlock (sometimes mistaken for parsley) could also be excluded. Diphtheria had been thought of only in connection with the first victim because of the paralysis of the eye and throat. But one condition remained for consideration, to wit, botulism. All had eaten of raw ham which had a bad odour and taste, and had on that account been given to the dog. Four members of the family who did not partake of this ham escaped the disease. Further, some of the ham had been cooked and eaten by one child who had not touched the raw ham. She remained well. One child may have owed his life to his school teacher who had told him never to eat ill-smelling meat. The dog, who ate between one and two pounds of the raw ham, was but slightly indisposed. Some of the meat remained and was variously tested. A filtered infusion poisoned mice without fail when simply eaten. Search was now made for bacilli, and the *B. botulinus* was obtained in pure culture. Botulism should be amenable to serum therapy, as it represents a pure toxicosis like diphtheria and tetanus, a fact which has long been recognised.—*Med. Record.*

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THE SIGNIFICANCE OF PAIN IN THE EAR.*

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PAIN in the ear is a symptom for which the practitioner is not infrequently consulted, and owns a multiplicity of causes. Upon early and correct diagnosis often depends the success of its treatment, since pain in the ear is as much a symptom of diseases of grave danger as it may be of conditions of trivial import. The non-recognition of its cause may, therefore, result in serious disaster, both immediate and remote, which might have been averted by prompt measures. I hope, therefore, to offer you to-night a short *resumé* of the causes which lead to pain in or about the ear, whereby you may more easily recognise them, and to this I shall add some hints upon treatment.

Pain may be referred by the patient to the ear, to the mastoid, or to parts of the head and neck around the ear. It may take any form, from one of agonising intensity to one of simple dull headache. Factors of grave importance in diagnosis are the possible accompaniments of constitutional disturbance, pyrexia, discharge, and vertigo, and when these are marked, they may render the symptom of pain alone one of secondary importance.

A classification of the causes leading to pain in and about the ear as a guide to differential diagnosis is best based upon its locality, and following this method, all forms belong to one of three groups, and the following list will be found, I think, fairly comprehensive:

(1) Pain in the ear.

(*a*) Without deafness or inflammation.

(*b*) With deafness, or inflammation, or both.

* Read before the Willesden and District Medical Society.

(2) Pain referred to the mastoid.
  (*a*) With local swelling.
  (*b*) Without local swelling.
(3) Pain outside the ear and mastoid.
  (*a*) Accompanying acute middle-ear suppuration.
  (*b*) Accompanying chronic middle-ear suppuration.

This arrangement tabulates conveniently the chief causes of pain in or about the ear, and we will consider each group in some detail.

Pain referred to the ear itself, when occurring without deafness or inflammation, is due usually to some reflex cause, such as the irritation of a carious tooth, disease of the tongue or tonsils. In the majority of cases the cause lies in the teeth, and a visit to the dentist cures the otalgia. It must be remembered that quite commonly there is no complaint of toothache at all. I once had quite a difficulty with a dental surgeon over one of these cases, in which he maintained that a recently filled tooth was not the cause of persistent pain referred to a perfectly normal ear, and he refused to extract it. He did so later, however, at the patient's own wish, but refusing to accept any responsibility. The tooth proved to be affected by periodontitis. At the hospital I have been several times consulted for ear pain due to early epithelioma of the tongue, the primary affection not having been noted by the patient, and once for otalgia due to carcinoma of the tonsil. I have also met with otalgia due to hypertrophied tonsils in a boy, æt. 13 years. Reflex pain in the ear is not uncommon also during the eruption of the last molar.

Occasionally people complain of neuralgia of the auricle, due, apparently, to pressure from lying upon it during sleep. It is at once relieved by wearing rubber air pads at night.

Pain accompanied by deafness or inflammation, or both, may be due to several causes. Occasionally foreign bodies or collections of cerumen give rise to pain, either in the ear or referred to regions outside. Such cases are quickly recognised upon examination.

It is convenient further to divide pain accompanied by deafness and inflammation according as the external or middle ear is affected. In the former class the impairment of hearing is generally moderate and follows the pain. It may be taken as a general rule that, when the pain is not severe, the meatus red, swollen and tender, its walls bathed with a slight amount of discharge, with fragments of macerated epithelium, and the introduction of a speculum is moderately painful, the condition is one of diffuse inflammation of the external meatus. I have seen several cases of this kind during the past few months of bad weather. They have occasionally followed influenza, and have sometimes been somewhat protracted by the desquamation of the tympanic membrane. The predominant micro-organism was streptococcus.

The symptoms of circumscribed otitis externa or aural furunculosis are much more marked. The pain is paroxysmal and severe, usually worse during the small hours of the night, and preventing sleep. The introduction of a speculum is attended with much pain and may be almost impossible. On inspection, one or two rounded swellings—boils—are seen in the meatus. Aural furunculosis is almost always heralded by itching and irritation in the meatus.

When pain is due to implication of the middle ear there is usually more or less deafness from the commencement. It may be due to simple acute inflammation of the tympanic membrane (so-called myringitis), to acute catarrhal otitis media, or to acute middle-ear suppuration. The points of diagnosis between the last two conditions often present difficulty in the early stages, and I shall, therefore, take myringitis first.

Acute inflammation of the tympanic membrane occurring *per se* is uncommon, although the condition is a frequent accompaniment of inflammation of both the tympanum and external meatus; it may, however, be caused by the entrance of cold water into the ear in bathing, etc., by draughts or by injury. When present, the pain (as can be readily understood when the fibrous structure of the membrana propria of the membrane is taken into consideration) is severe, deep-seated, tensive in character, accompanied by throbbing and tinnitus and much aggravated by external sounds. Inflation increases both the pain and deafness. The impairment of hearing is usually moderate and there is intolerance of noise. On inspection, the membrane is seen to be much reddened. In the early stages engorged vessels can be made out, chiefly along the handle of the malleus, but these are soon obscured by the diffuse reddening and

swelling which quickly follows. The usual landmarks become indistinct to obliteration and the whole surface of the drumhead becomes lustreless and blueish-red.

It is impossible to draw any hard and fast line between simple and suppurative acute middle-ear inflammation, since the two forms differ only in their intensity and mode of attack. To commence with, the vessels of the tympanic mucosa are injected, the mucous membrane swollen, and a serous exudate, later becoming mucous, occurs. If the inflammation progresses, the mucous swelling is intensified and pus forms. It is easy to appreciate how, in a cavity with unyielding bony walls, like the tympanum, tension may quickly become marked. It must be borne in mind, therefore, that "acute catarrh" and "acute purulent inflammation" of the middle ear are but degrees of the same process, and that treatment at the outset must be directed to the same end—the control of the process and the prevention of the milder degree from running on to the more severe. The character of the invading organisms also has a decided influence upon the intensity of the inflammatory process.

Acute middle-ear inflammation usually begins with sudden pain, ushering in a sense of fulness and obstruction. Tinnitus, either hissing, rushing, or throbbing, quickly follows, the pulsations being synchronous with the pulse. In the early stages the hearing is but slightly impaired, but as exudation takes place the deafness becomes considerable, a point which distinguishes the condition from uncomplicated myringitis. Autophony, or the hearing of one's voice with extreme loudness, as if one's head were shut up in a box, is a common symptom. Pain is violent, and much worse at night, radiating over the side of the head and intensified by movement of the jaw. There is considerable constitutional disturbance, with temperature, especially in children, in whom, too, the mastoid antrum often shares in the inflammation. When the condition does not pass beyond the simple catarrhal type these symptoms soon reach their height, remain thereat for twenty-four or forty-eight hours, and then disappear quickly. In very slight cases the condition may be limited to simple hyperæmia without exudation, merely causing moderate deafness, with pain and tinnitus. These transient recurrent attacks of earache are common in children, and I would impress upon you the fact that they should never be neglected, being almost, if not always, due to infection *viâ* the Eustachian tubes from adenoids. They are very often the forerunners of chronic catarrhal deafness in young adult life, in which there is often a history of attacks of earache in childhood, and it is during the latter period that prompt recognition and treatment of the cause may save the victim from severe deafness in later years.

In more severe cases of acute tympanic catarrh, tension increases sufficiently to cause perforation of the membrane, with discharge of bloody serum, subsequently giving place to mucus. This perforation relieves tension, the symptoms abate, discharge ceases, and the membrane quickly heals.

When the inflammation is suppurative it begins very similarly, but the symptoms are often more severe at the outset, and if not, rapidly become so. Pain, the most prominent manifestation, is intense and continuous, tinnitus is intolerably violent, and the pulsating arteries give the sufferer the sensation of a hammer and anvil at full work in the head. The pain is always worse at night and the patient is pulled down by broken rest. There is great constitutional disturbance, with fever, often high, and frequently accompanied by rigors. Vertigo, delirium and cerebral symptoms may appear, and in children (sometimes also in adults) the condition may be mistaken for meningitis. Sooner or later perforation occurs, and with the appearance of the purulent discharge the severity of the symptoms abates. Temperature falls, any cerebral symptoms disappear, pain and tinnitus lessen.

The appearances of acute middle-ear inflammation are usually those of myringitis, the membrane always partaking in the inflammatory process. The most reddened portion is generally the posterior superior part, and there may be bulging from the exudate in the tympanum. The most common spot for perforation to recur is in the anterior inferior quadrant. When suppuration has already taken place the bulging may present a yellowish reflex. If perforation has occurred recently the swelling and discharge often obscure the view, but the site of the hole can generally be recognised by a pulsating light reflex on the fluid lying over it.

In the early stages of acute inflammation of the middle ear it is practically impossible to say

with certainty whether it will assume a catarrhal or purulent character, or whether the attack will be mild or severe. Fortunately, however, the treatment will be the same, and therefore it is not absolutely necessary to arrive at a definite conclusion. There are, however, one or two indications by which one may be enabled to form some opinion as to the probable course of the case. When the redness seen in the drumhead is limited to Shrapnell's membrane, the inflammation is likely to be purulent in form and severe in type. Other signs having a similar indication are severity of local and constitutional symptoms. When bulging of the membrane has appeared, severe and purulent inflammation may be expected if the swelling is limited to the posterior superior quadrant, especially if it shows a yellowish reflex. Another indication is sometimes furnished by the severity of the nasal or throat inflammation from which the primary infection of the ear started.

Passing now to pain referred to the mastoid, this, when accompanied by local swelling, may be due to one of three conditions: inflammation of a mastoid lymphatic gland, mastoid periostitis, cortical mastoiditis.

The first of these may be dismissed at once. The swelling is circumscribed and movable, and the pain is not great. The mastoid glands receive their lymphatics from the posterior parietal region and the back of the pinna and the cause of their implication must be sought in those regions.

Mastoid periostitis is due as a rule to disease in the external meatus, or it may occur by the spread of inflammation from the middle ear by the fibrous tissue connecting the mucous membrane of the tympanum with that of the meatus, or by the blood-vessels. In children additional facilities exist in the unclosed petro-mastoid suture and the large mastoid emissary vein. The pain has the characteristics of a periostitis, and is worse on pressure. The swelling is immovable, shelving down to the bone, and later becoming somewhat boggy. The auricle becomes displaced downwards, forwards and outwards, standing out prominently from the head.

Cortical mastoiditis, a condition of suppuration in the superficial cells of the mastoid, is a result of acute middle-ear suppuration. The swelling is obviously bony, and develops rapidly. It is not of frequent occurrence.

More serious than the conditions just mentioned is acute deep mastoiditis. This may be a complication either of acute or chronic middle-ear suppuration, and calls for prompt relief. Pain is of a deep, throbbing, boring character. Cases of comparatively painless acute mastoid disease have been recorded, but they are the exception. The symptoms due to pus in the mastoid antrum are chiefly pain, increased by pressure or percussion. Unless relieved, the tension within the mastoid increases and there is rise of temperature with concomitant symptoms. The aural speculum shows the membrane to be swollen and congested and the landmarks blurred. The seat of any perforation is generally marked by pulsating fluid, and discharge may rapidly accumulate after removal owing to overflow from the pus-filled antrum. A most important sign and one which is often considered to be pathognomonic is bulging of the superior posterior meatal wall, close to the membrane. You will note that I have said nothing about redness and swelling or œdema over the mastoid. It may occur, and it may be there when you are called in, but it should be clearly understood that the case ought never to be allowed to reach that stage, which means that the empyema of the mastoid antrum has reached the surface and has either perforated the bone or is on the point of doing so. Still, some very acute cases are so rapid that swelling and œdema may be a comparatively early condition, and such cases admit of no delay in operation. I have seen, in influenzal cases, the whole mastoid destroyed in two days, but such cases are less common than they used to be. One other point worth knowing in dealing with these acute inflammatory swellings over the mastoid: they may be due to meatal inflammation, and in such cases the post-auricular fold becomes obliterated. This obliteration never occurs when the swelling is that of mastoid disease; this is a useful diagnostic point. Another point is that temperature is, in my experience, less to be relied upon as a symptom than are the other objective signs of which I have just spoken; this is less true in children, perhaps, although even in them it is not a symptom upon which much dependence is to be placed.

It would be beyond the limits of this paper were I to attempt a detailed discussion of mastoid conditions, and I must, therefore, be content with

mentioning the character of the pain when mastoiditis is a complication of chronic suppuration. As a rule, the subjective signs of chronic mastoid empyema are comparatively insignificant, pain being marked only when an acute exacerbation occurs upon an already existing chronic condition (due either to blocking of the outlet into the tympanum or to a fresh infection), or when caries of the mastoid walls has started an intra-cranial complication. The objective signs are, however, of great importance, and may be all or some of the following: More or less copious discharge, usually fœtid from retention, and at times sanguineous; granulations, especially when growing from the region of the posterior part of the attic, and which recur quickly after removal; enlarged glands round the ear and beneath the pinna; facial paralysis occurring during the course of a discharge of long standing; bulging of the posterior superior meatal wall, and the presence of carious foci.

I would pause here for a moment to point out that the anatomical peculiarity of the mastoid process very frequently, if not always, determines the course of a middle-ear suppuration. It is the large-celled pneumatic variety which, in an acute middle-ear suppuration, becomes so readily infected. Looking through the notes of a considerable number of operations for acute mastoid empyema, I find that 90 per cent. were of this variety, and of the cases that followed influenza the percentage was 98. The opposite variety of mastoid, in which the antrum is small, deep, and situated in a mastoid process of dense or slightly diploëtic bone —the so-called "infantile" variety of Cheatle—is almost invariably found in chronic middle-ear suppuration, and is the type which one meets with in cases where intra-cranial complications have arisen in the course of a chronic otorrhœa.

I now pass to the consideration of pain occurring outside the ear and mastoid process, a class containing complications of ear disease which are most dangerous to the patient and difficult to the surgeon. They are those accompanied by severe constitutional disturbance and derangement of the cerebral functions. Time will, however, permit only of a brief indication of the complications which may be expected and the method of detecting them.

When pain is diffused about the ear in the course of an acute middle-ear suppuration and is of moderate severity it may be due merely to the tension of retained pus, and it is but a part of the pain in the ear itself. It will be relieved by that valuable little operation, incision of the tympanic membrane, to which I shall refer again, with or without washing out of the tympanum. But this diffused pain may also be the beginning of the most fatal of all intra-cranial complications—meningitis. In such cases the importance of early diagnosis cannot be over-estimated. Uncomplicated meningitis is as frequently met with in cases of acute suppuration as in cases of chronic disease in children, but less frequently in adults, the channels of infection being more patent in early life. In from 70 to 75 per cent. of the cases the meningeal infection starts in the posterior cranial fossa, in from 25 to 30 per cent. in the middle fossa.

The symptoms of a commencing meningitis are often very vague, localised headache on the side of the offending ear being the most constant and pronounced, subsequently and often rapidly spreading over the head. It is aggravated by light, noise and movement of the head. In early stages vertigo is not infrequent and constipation is usually marked. Delirium, high temperature, retraction of the head, Kernig's sign, severe retching and vomiting are other symptoms. Optic neuritis and choked disc are not to be depended upon for diagnosis, and I think that when, in the course of an acute middle-ear suppuration, there occur severe headache with constipation, Kernig's sign and retraction of the head, especially with delirium in young children, the case is sufficiently suspicious to warrant immediate lumbar puncture and microscopical examination of the cerebro-spinal fluid.

In chronic suppuration of the middle ear a different set of complications is presented. Of these, four may be enumerated, all of them formidable. The patient may complain of occasional attacks of localised headache about the mastoid region. Such complaint should always arouse suspicion as to the presence of chronic deep mastoiditis, in which the occurrence of headache is due to th hygroscopic swelling of collections of cholesteatomatous material. The presence of small, white masses of dead skin in the middle ear on inspection will serve to strengthen this suspicion. Unless dealt with surgically, the cases may easily pass on to caries, thrombosis of the sigmoid sinus, or cerebral abscess.

Another complication, less easy of diagnosis, is that of extra-dural abscess. It may be suspected if there is continuous high temperature, with localised swelling or tenderness on percussion, mental irritability, dizziness, nausea and vomiting. If unrelieved, symptoms of cerebral compression may make their appearance. Symptoms may further be modified by the locality of the abscess: when about the tip of the petrous bone, paralysis of the sixth nerve is common; when in the sigmoid groove, the case may be complicated with signs of sinus thrombosis, possibly with those of cerebellar abscess.

A third and very serious complication is that of sigmoid sinus thrombosis. Its symptoms are largely those of the septic intoxication to which it gives rise. It may come on gradually or very suddenly, and a rapid rise of temperature, with rigors, in the course of a chronic suppuration is very suggestive of a commencing septic phlebitis. From normal, the temperature may shoot up to 102°, 103°, or more, and drop as suddenly, with profuse sweating, to normal or subnormal. When uncomplicated, the pulse-rate corresponds with the temperature. Pain is almost always present over the mastoid, the sinus and down the neck, persistent, severe, and intensified by pressure and percussion. It is sometimes accompanied by torticollis. Optic neuritis occurs in about 40 per cent. of cases. Œdema over the mastoid and down the course of the internal jugular vein may be present.

Fourthly, there is the complication of cerebral or cerebellar abscess. In cephalic abscess, dull, localised headache, increased by pressure and percussion, is usually one of the earliest symptoms. Another usually early symptom is cerebral vomiting. Slow cerebration, subnormal temperature, pulse of cerebral character and other pressure symptoms are common. There is no need to go into the differentiation of tempero-sphenoidal and cerebellar abscess here, as I merely wish to give indications whereby a cephalic abscess may by suspected.

These, gentlemen, are the most notable of the causes of pain in and about the ear, and I trust I have made clear to you some of the characteristics by which they may be diagnosed. Pain due to malignant disease I will merely mention, for the chief cause of carcinoma of the ear is the irritation of long-continued suppuration, and primary sarcoma is uncommon. Let us now turn our attention to treatment.

In discussing treatment we may at once dismiss that of the intra-cranial complications with which the past few minutes have been occupied. Nothing short of operation (and at the present day even meningitis, if caught early, is not altogether hopeless) will suffice, and I take it that it is rather with their diagnosis that we have most to do. Therefore we will leave the consideration of the treatment of meningitis, cerebral and cerebellar abscess, extra-dural abscess and sinus thrombosis, and occupy ourselves with that of other causes of pain.

What has been said of the causes of reflex otalgia gives the indication of its appropriate treatment. The existing trouble—a carious tooth, disease of tongue, fauces or pharynx—must be sought for and removed, or otherwise appropriately treated. Similarly, foreign bodies or collections of cerumen causing pain give equally clear indications.

In considering the treatment of external otitis, the two forms—diffuse and circumscribed—must be taken together, since, to some extent, the lines upon which it is conducted are the same. Everything that promotes congestion of the head and ear must be guarded against, and the congestion present relieved. The most valuable agent at our disposal is local depletion by leeches, natural or artificial. One or more leeches should be applied first in front of the tragus, that being the spot where the veins conveying the blood from the ear emerge. Before applying them, the meatus should be closed by a plug of wool.

Another very valuable method is the use of heat or cold. The former may be applied by means of hot instillations of sterilised water, as hot as can be borne, or by a pledget of cotton-wool steeped in hot water, hot boric lotion, or hot Goulard water. Poultices are to be avoided, as they are apt to cause perichondritis of the auricle, besides the fact that their continued use induces an increased blood-supply to the whole ear.

Cold may be applied by an ice-bag, or by Leiter's tubes. Some cases are much relieved by a combination of heat and cold, the latter round the ear, the former by hot instillations in the meatus.

Sedatives applied to the meatus afford another means of relief. They may be used as instillations, such as cocaine 2 to 5 per cent. combined with 1 per cent. of morphia, or by Gruber's "aural

ovoids," small cones of gelatine, combined with various drugs designed for introduction into the meatus. In both diffuse and circumscribed external otitis I have lately found that a solution of anæsthesin gr. xv, absolute alcohol ʒiiss, liquor aluminis acetatis ɱxxx, and glycerine ʒj is useful. A small piece of ribbon gauze is soaked in this mixture, introduced into the meatus for twenty-four hours and kept moist by dropping in the solution from time to time.

In the circumscribed form—aural furunculosis—additional treatment is necessary, and thorough antisepsis is of great importance. The meatus should be gently purified with perchloride solution (1 in 2000), or with Lister's strong mixture, and the furuncle opened as soon as its site is detected. This is a very painful procedure, and is best done under one of the short anæsthetics. A very sharp tenotome, or better still, Grant's special furuncle knife, is needed. The boil must be incised through its centre, and the meatus packed with ribbon gauze soaked in an antiseptic. After-treatment should be by warm instillations of strong spirit twice a day.

In both forms of external otitis, constitutional treatment, started by a brisk purge, is necessary.

In dealing with aural furuncles it must be remembered that they are due to staphylococci, and that repeated reinfection resulting in crops of boils is the rule. Spirit instillations should be kept up twice daily for at least a week after all symptoms have disappeared. A practical point is that accumulations of dead skin and cerumen form a harbour for the organisms, and that they must be cleared out to prevent reinfection. Occasionally boils recur repeatedly in spite of treatment, and in such cases an autogenous vaccine is often of value.

The treatment of pain resulting from acute otitis media must be both local and general. Strictly speaking, the methods at our disposal may be divided into three groups: prophylactic, abortive, and operative. Of these I shall merely mention the first, as we are dealing with the treatment of pain, that is, with a condition that has already become established. In parenthesis, however, I may say that a long experience has taught me that numbers of these cases could be prevented by timely treatment of the routes by which the ear becomes infected, viz. the upper air-passages.

When an attack of acute middle-ear inflammation is established prompt treatment is necessary. The matter of first importance is absolute rest. The patient should be confined to his room, and if there is a likelihood of the case assuming the suppurative type, he should be kept in bed; stimulating foods, alcohol, mental excitement and physical exertion must be absolutely forbidden. A purge should be given, preferably of calomel, followed by a saline. Insomnia may have to be combated by opiates, but the best method of procuring sleep is to allay the pain. Heat or cold, or a combination of the two, is useful, but, as a general rule, heat, especially dry heat, is preferable to the patient. Hartmann, pointing out that the continued influence of heat kept the ear in a condition of congestion which retarded resolution, whilst cold around the ear lessened the inflammation, suggested the combined action of the two agents. By his method, which is certainly useful, to the region round the auricle cold is applied by ice-bag or Leiter's tubes, and hot fluids (115° F.) will suffice) instilled into the meatus.

But the best method of all is the application of leeches. From two to six, applied over the mastoid and in front of the tragus, will often rapidly cut short the pain and inflammation. Their application should be followed by dry heat by means of hot flannel, or wool, or a bran-bag.

When the condition is subsiding, inflation is useful, as it restores hearing more quickly, and by improving the ventilation and drainage of the middle ear, accelerates recovery. It is, of course, best done by means of the Eustachian catheter, but if the Politzer bag is employed, the nasal passages should be cleansed preparatory to its use.

In the early stages I have sometimes been able to abort an acute otitis media by means of catheterisation several times repeated during the day.

It is important to treat the upper air-passages concurrently when trying to abort an attack, as by this means the ear is reached by two routes—meatus and Eustachian tube. During the past year the use of the Holmes' naso-pharyngoscope has shown one that in all these cases there is much swelling and tumefaction about the pharyngeal mouth of the Eustachian tube. Frequent inhalations of steam impregnated with Friar's balsam or menthol are most useful in this respect.

In a certain number of cases abortive measures

on the lines just described will be sufficient to gain our object, especially if the case be one of the milder, catarrhal type. Unfortunately, a certain percentage will require further measures. It is unwise to wait too long and to continue to employ abortive treatment when it makes no impression. If the symptoms continue unabated for twelve hours, or if, whilst they diminish under treatment during the day, they return with their former or increased severity during the night, then it is imperative that something further should be done. The middle ear is in a condition of severe tension, and this must be relieved if you do not wish to risk serious damage to the hearing.

The operative treatment which then becomes necessary is simple. It consists in freely incising the tympanic membrane—a procedure sometimes called "paracentesis" or "myringotomy," but which is better named simply "incision." By incising the drumhead much damage to the ear may be prevented, and, since wounds of the tympanic membrane heal very quickly, it is far better to incise unnecessarily than to leave things too long. It must be remembered that an acute otitis media practically means an inflammation of the whole middle-ear track, in which Eustachian tube, attic, tympanum and mastoid antrum all share, and are all in a condition of more or less tension. If the membrane is allowed to perforate spontaneously the tension is continued over an unnecessarily long period, and the damage done to the drumhead is far more than that resulting from a clean surgical incision. Prompt operation, therefore, can do no harm, whilst its neglect may mean acute mastoid complications or a chronic discharge, with all its attendant troubles and possibilities. If, on inspection, the membrane is seen to be bulging, there is no possible reason for delaying incision, and when the surgeon has any doubt as to the propriety of the proceeding, he will find it the best and wisest course to incise.

As regards the *modus operandi*, there is not much to be said. Many special instruments have been devised, but the best is a fine tenotome or a Sexton's knife. This should be as sharp as possible. Grant has devised a shouldered knife which is useful for those who are diffident as to their skill and fear to cut too deeply.

As a preliminary, the meatus must be thoroughly purified by swabbing with perchloride solution, Lister's strong mixture, or rectified spirit. The incision, which is very painful, although the pain is but momentary, may be made under gas or a local anæsthetic. The best form of local anæsthesia I know, short of the infiltration method, is that attained by placing a small plug of wool, steeped in a mixture of equal parts of cocaine, menthol and pure carbolic acid, against the drumhead for ten minutes. The head requires to be kept steady, and the incision is made under the best illumination obtainable. If the membrane be bulging uniformly, incise in the posterior inferior quadrant; if only a part bulges, cut through the maximum bulge. The incision should be bold and free, not a mere puncture, which is worse than useless, for the patient only has to have it done a second time. After the incision, introduce a piece of ribbon gauze with aural forceps into the meatus and make sure its end is carried right up to the incision. Leave this, as a drain, for twenty-four hours, continuing to use dry heat to the ear to assist further to allay the pain. Syringing or irrigation is unnecessary to remove the secretion, unless the latter is very tenacious. Politzerisation as a means of blowing secretion out of the incision should not be practised, as, by blowing micro-organisms from the naso-pharynx—almost always the original source of the infection—a simple case may be converted into a suppurative one, or, if pus be formed already, it may be driven by the inflation into the attic or the mastoid antrum.

In the majority of cases of acute middle-ear inflammation thus treated, relief is permanent with the cessation of tension, and discharge disappears in a day or two.

When acute suppuration of the middle ear is accompanied by mastoiditis, treatment should be on the lines just given until the question of operation has been decided upon—namely, incision of the membrane, the application of leeches and heat, together with constitutional measures.

As regards the treatment of mastoid disease, it will scarcely be fitting to discuss here the question of the mastoid operation. Probably, as I have already pointed out, every case of acute middle-ear inflammation has the mastoid antrum involved to some extent. Therefore the avoidance of Politzerisation or syringing after incision of the membrane has been performed is important, in order that the possibility of secondary infection of the mastoid

antrum may be reduced to a minimum. The treatment of acute middle-ear disease must be prompt, so as to give the condition less chance of spreading, and only by watchfulness and readiness can you help your patients to avoid its serious complications.

In an earlier part of this paper I referred to the prophylactic treatment of acute middle-ear conditions as being outside the scope of my remarks. I would, however, like to make some allusion to it in conclusion. When you have treated one of these cases and brought it to a successful issue, overhaul the patient with a view to finding out the cause of his recent trouble so that he may be saved any further attacks. You will most likely find it in the upper respiratory passages. If the patient is a child, it will most probably be in the naso-pharynx. It is not infrequently found that, even after the removal of adenoids, apparently successfully, a child has an attack (recurrent or primary) of acute middle-ear suppuration. The reason is either that the adenoids have not been removed completely, or that the after-treatment of the operation has been neglected. In the former case one usually finds that, although a large central mass has been well excised, the recesses behind the Eustachian openings—Rosenmüller's fossæ—have not been cleared, and it is from these regions that the ear becomes infected or reinfected by way of the Eustachian tube. In the second case—neglect of post-operative treatment—in 75 per cent. of adenoid cases there is a pharyngitis or rhinitis left by the adenoids, and, unless this is treated by appropriate methods, a source of infection will remain in the naso-pharynx. When one considers that the naso-pharynx is a sort of anatomical meeting of the ways, its importance as a centre of infection is realised.

March 31st, 1913.

HUMAN PHYSIOLOGY. By Prof. Luciani. Translated by Miss F. E. A. Welby. Edited by Dr. M. Camis. Vol. II. Macmillan & Co., 1913. Price 18/- net. To be completed in 4 volumes.

THE profession in this country is indebted to Miss Welby for the translation from Italian of this excellent, well-arranged, and comprehensive text-book of physiology. The present volume deals with the internal secretions, digestion, excretion, and the skin.

TWO CLINICAL LECTURES ON CALCULUS IN THE UPPER URINARY TRACT.*

By F. J. STEWARD, M.S., F.R.C.S.,
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II. URETERAL CALCULUS.

WHEN a stone passes from the pelvis of the kidney into the ureter, subsequent events are determined by its size; for a very small stone will offer but little resistance to the contractions of the ureter, and hence will rapidly pass down into the bladder. Such an easy transit is accompanied by no pain at all in some cases, and in others a transient attack of pain occurs which is quickly forgotten by the patient, although it may be sharp while it lasts. I mentioned in my last lecture the case of a woman from the pelvis of whose kidney I removed over 100 small stones. She came to the hospital on account of frequent attacks of pain in the right side of the abdomen, which lasted only a few minutes, and which were unaccompanied by any hæmaturia or other sign of renal calculus. The patient was told to examine her urine after the attacks of pain, and she shortly returned to the hospital with several tiny stones that she had recovered from her urine.

In children, again, small stones pass down the ureter without their being aware of it, and either form the nuclei for vesical calculi or subsequently become impacted in the urethra.

A larger stone gives more trouble to the ureter, and excites violent contractions of the tube, which find expression in an attack of more or less severe renal colic, the pain suddenly ending as the stone drops into the bladder. In such a case the mucous membrane of the ureter is usually torn by the stone, hence the appearance of hæmaturia after an attack of renal colic. As the size of the stone further increases, it is clear that a point will be reached at which the power of the muscular contractions of the ureter become insufficient to force it into the bladder, so that it becomes impacted in the ureter, and it is with such cases that this lecture is concerned.

* At Guy's Hospital.

Now, the lumen of the ureter is not uniform throughout, being narrowest near the entrance into the bladder, and also constricted to some extent where it crosses the brim of the pelvis, so that *à priori* one would expect a calculus impacted in the ureter to become so impacted either at the constriction at the pelvic brim, or, should it be small enough to pass this point, at the narrow part near the bladder. This expectation is, in fact, borne out by clinical experience, the majority of impacted calculi being found in the last inch of the ureter, the rest at the brim of the pelvis.

Now let us consider the symptoms caused by the impaction of a calculus in the ureter. In nearly all cases, as you would expect, the first event is an attack of renal colic, accompanied, as is usual, by hæmaturia. This attack of colic does not, however, end in the typical way—*i. e.* suddenly—but gradually dies away, no doubt owing to the muscle of the ureter being gradually worn out, only to recur from time to time, usually in less severe degree, as the ureter recovers sufficiently to renew its efforts at expulsion.

In a few cases, however, there is no such initial attack of renal colic, and this perhaps particularly applies to children. For instance, I have removed a stone from the lower end of the ureter of a boy, æt. 8 years, which had apparently caused no symptoms at all, for the stone was discovered accidentally by Mr. Ironside Bruce when he took a radiograph to show another stone that lay in the boy's bladder.

In rare instances adults exhibit the same absence of pain. For instance, in the case of Mrs. T—, to which I shall refer again later, the stone which I removed from the lower end of the right ureter had reached that spot without any pain at all, although she had frequent attacks of hæmaturia.

The further results are determined by two circumstances — the proximity of the point of impaction to the bladder, and the size and shape of the stone.

If a stone becomes impacted low down in the ureter, and quite near to the bladder, it is a curious fact that the symptoms usually present are those of bladder disease, such as pain or irritation referred to the neck of the bladder or end of the penis, frequency of micturition, hæmaturia, and pyuria.

It is important to note this feature carefully, for as you will observe the symptoms are very deceptive, being those commonly associated with stone in the bladder, and hence a stone in the ureter may easily be missed altogether if the bladder only is suspected and examined with a negative result The following two cases illustrate these points very well.

Case 1.—Mrs. T— was sent to me by Dr. McPherson in May, 1911. Her history was as follows: During the previous three months she had had frequent attacks of hæmaturia, associated with a feeling of irritation not amounting to pain referred to the region of the neck of the bladder. The attacks occurred about once a week with absolute suddenness, the urine being quite red from blood after having been perfectly clear perhaps only an hour previously. She had some frequency of micturition, and with this the feeling of irritation, but she assured me that she had had no pain at any time.

On examining the urine I found it acid, free from blood, but containing a considerable amount of pus.

Abdominal palpation revealed the fact that the right kidney was slightly enlarged, but not at all tender.

An X-ray examination was then made, and the radiogram showed very clearly a small, irregular stone impacted in the lower part of the right ureter. The question that now arose was whether it would be wise to wait awhile to see if the stone would pass into the bladder. As, however, the symptoms had already persisted for a considerable time, and as, moreover, the obstruction to the ureter was causing damage to the kidney, as shown by its enlargement and the presence of pus in the urine, I deemed it safest to remove the stone without delay, lest further harm should come to the kidney. This was done by the following method, which I always adopt in these cases, and which has been uniformly successful. Through a somewhat curved iliac incision the abdomen is opened. The stone having been located, pressure is now exerted upon it so as to force it up the ureter to a point above the brim of the pelvis. This can almost always be done, as the ureter is dilated behind the stone. The incision in the peritoneum is next closed, and the parietal peritoneum stripped inwards until the ureter is reached. Having then manipulated the stone into the most convenient part of the ureter, this is isolated with gauze and

clamped above the stone to prevent soiling of the wound by urine and pus when the ureter is opened. This is done in a longitudinal direction, and the stone removed, the incision in the ureter being then closed by fine catgut sutures passing through the outer coats only. The clamp is then removed and the parietal incision closed. The patient made an interrupted recovery and returned home on the twenty-first day, the urine, which for a few days after the operation contained pus, being then normal, and the kidney no longer enlarged.

CASE 2.—The next case exhibits similar bladder symptoms. The patient, a man, æt. 24 years, was admitted under my care into Guy's Hospital, for painful micturition and hæmaturia. On several occasions during the last three years the patient had slight attacks of painful micturition, but these had always passed off in a few days without treatment. Latterly, however, the pain had become more frequent, and also more severe.

These attacks were always accompanied by increased frequency of micturition, and also by the passage of a small amount of blood with the urine. The patient felt quite certain that he had never passed any stones.

A week before admission pain became much more severe, and the urine became scarlet in colour from the presence of blood, and continued so until admission into the hospital. On admission the urine was acid, had a specific gravity of 1025, was quite red from the presence of blood, and also contained a small amount of pus. Micturition was accompanied by severe pain both in the perinæum and also at the end of the penis. There was also pain at the end of the penis quite apart from micturition, but increased by micturition and also by taking exercise. The severity of the pain, the patient said, was such that he had been obliged to give up his work and seek advice. Micturition was also not only frequent but difficult, for he made frequent attempts without any result. Nothing could be made out by examination of the abdomen or rectum, and there was absolutely no tenderness or fulness over either kidney. It was thought that there was probably a stone in the bladder, so a sound was passed, but nothing could be felt.

On two occasions, moreover, the patient was radiographed with a negative result, so I was forced to the conclusion that the cause of the symptoms was in the bladder, which I therefore explored through a supra-pubic incision. I found nothing amiss with bladder, but through its posterior wall I felt a small stone in the right ureter. This was removed as in Case 1, and the patient was cured.

Turning now to the other factor in the results of impaction of a stone in the ureter, namely the size and shape of the stone, it is evident that any stone must in some degree cause obstruction with consequent damage to the kidney. Small irregular stones, such as were present in Cases 1 and 2, do not, as a rule, cause marked obstruction, so that secondary kidney changes are slow, but if the stone does not pass, the inevitable terminal result is destruction of the kidney with the formation of a hydro- or pyo-nephrosis, and it is this fact that renders the early diagnosis and treatment of ureteral calculus so important.

A larger stone will necessarily produce a greater degree of obstruction, which may even be complete. In such case the kidney will quickly be destroyed. and should the opposite kidney be absent or destroyed by previous disease the result will be anuria, which will be fatal unless immediately relieved by operation.

If the obstruction is, however, severe but not complete, a large hydronephrosis may form as in the following case, and the patient come under observation on account of the tumour so caused.

CASE 3.—The patient, a woman, æt. 42 years, was admitted under my care into Guy's Hospital in January, 1910, for a tumour on the left side of the abdomen. She gave the following history. Five weeks previously she was seized with severe pain in the left loin shooting down into the groin. After some hours the pain gradually subsided into a dull ache, which entirely passed off in twenty-four hours. (Note that the pain subsided gradually, not suddenly as it does when a stone either passes into the bladder or drops back into the pelvis of the kidney.)

After a day or two the pain returned for a time, and continued in intermittent attacks for a fortnight, but for the three weeks preceding admission to hospital she had had no pain at all. During this time, however, she had noticed swelling of the abdomen. On admission a large fluid tumour was found occupying the left side of the abdomen, reaching back into the loin and forwards to the umbilicus.

X-ray examination revealed a large calculus in the ureter, and this could also be felt *per vaginam*.

I removed the stone as in the other cases, and the wound healed at once without any leakage.

After the operation a large amount of purulent urine was passed, and the tumour gradually subsided, so that when the patient left the hospital a month later it could only be felt with difficulty, and the urine was free from pus.

The results of operation for ureteral calculus are, as you will have gathered from the cases I have described to you, eminently satisfactory, the striking feature being, perhaps, the fact that so small a tube as the ureter heals so readily without leakage if properly sutured, for in a considerable number of these operations I have not had a single case in which any leakage has occurred from the sutured ureter.

March 31st, 1913.

ETHYL CHLORIDE ANÆSTHESIA.

A Lecture delivered at the North-East London Post-Graduate College.

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BOTH in this hospital and in private work I have been accustomed for several years to induce anæsthesia by means of ethyl chloride as a routine method. In doing so I have often found that medical men look upon the drug with great distrust or profess entire ignorance as to its use. For this reason it may be of interest to give a short account of ethyl chloride anæsthesia, as I think it an anæsthetic of very real value and one which may be given safely by any competent person.

Ethyl chloride or kelene is, as its name implies, C_2H_5Cl. It is prepared by acting on ethyl alcohol by hydrochloric acid gas. It is a clear liquid with an aromatic and rather pleasant odour and boils at 54·5° F. This is important, as it means that the fluid must be kept in closed containers, and it also ensures its rapid vaporisation at the temperature of an operating theatre or even in a colder room when the container is held in the warm hand. The vapour is nearly two and a half times as heavy as air. This also is of practical importance, as I have sometimes found that when the induction of anæsthesia is unusually slow it may be hastened by agitating the bag of the inhaler. This suggests that the heavy vapour may tend sometimes to lie in the lower part of the bag.

Ethyl chloride is made by several firms. In 1905 the 'Lancet' reported that the products made by Duncan & Flockhart, Hedley and Kuhn were absolutely pure, as also was that sold under the name of "kelene." Personally I have almost always used that manufactured by the two first-named firms, and I have found them both excellent.

I need not trouble you with many details of the history of the anæsthetic. As long ago as 1847 Flourens discovered its anæsthetic properties, and the very next year Heyfelder used it with success on his patients. For some reason it seems to have been no further used for a long time, though in 1880 a Committee of the British Medical Association gave an unfavourable account of it, as they reported that it caused convulsions and stopped respiration. Owing to its power of rapid evaporation it has of course long been used as a local anæsthetic. Its use as a general anæsthetic was apparently revived by the accident of two dental surgeons independently discovering its power while using it for local purposes. A distinguished surgeon now connected with this hospital also made the same discovery while freezing some small nævus in the vicinity of the nose. As the patient happened to be a very close connection of his own his grave alarm prevented him from appreciating the scientific value of his observation.

In 1896 Lothiesen tried ethyl chloride in 170 cases, using a Breuer's mask. This was, in effect, an open method, and did not allow re-breathing. He records no untoward result, but the anæsthesia seems to have been rather uncertain and always very light.

However, it is really to McCardie that the present general use of the anæsthetic in this country is due, and most of our knowledge of its history is also drawn from his papers. In 1901 he published a few cases given by the open method with fair success only. However, in 1903 he gave an account of 250 cases in which he had administered the drug by a closed method, namely, in an Ormsby's inhaler, with very satisfactory results. The next year Hewitt made a series of experiments at St. George's Hospital on the relative advantages of the open and closed methods and decided emphatically in favour of the latter. Since that time the use of the anæsthetic by some closed method has become very general, and I cannot find that any very great advance has been made except that a number of new inhalers have been described and illustrated in the medical journals.

Ethyl chloride may be used as a general anæsthetic in three different ways. Firstly, much as nitrous oxide is used, to produce anæsthesia of short duration for rapid operations. Secondly, again, like nitrous oxide, to induce anæsthesia quickly preparatory to carrying on with chloroform or ether. Thirdly, for maintaining anæsthesia for half an hour or more without the use of any other anæsthetic. For this last purpose I do not consider that ethyl chloride is a very suitable drug unless under special circumstances, and I am sure that prolonged anæsthesia under its action should not be attempted by anyone who is not thoroughly familiar with the technique.

Now as to apparatus. In children, who take this anæsthetic particularly well, good results can be obtained by spraying it on to a Skinner's mask. However, the use of a closed bag is now very general. At one time I always used an inhaler devised by Mr. Charles Heath. For this one obtains the ethyl chloride in sealed tubes containing either 3 c c. or 5 c.c. By an ingenious arrangement the neck of the tube is broken by moving a lever, which simultaneously closes the air opening and the ethyl chloride escapes into the bag. The method is satisfactory, and has the advantage that none of the vapour is lost. On the other hand the tubes are somewhat expensive, and the breaking of the glass with the resulting explosive sound is apt to be disturbing to a nervous patient. Also if the original charge does not prove sufficient it is rather awkward to have to remove the empty tube and insert a fresh one. This difficulty I always avoided by placing a spare tube in the bag itself, and if necessary snapping the neck with the fingers through the indiarubber.

I now always use the "spurt method." One of the best inhalers for the purpose is the so-called Simplex inhaler introduced by Dr. Luke. The ethyl chloride is obtained in graduated glass tubes usually holding 60 c.c., and fitted with a special nozzle by which any desired quantity of the fluid can be injected into the bag through an opening made for the purpose. This opening is then at once closed by a metal ball held in position by two springs. I find the special nozzle supplied by Messrs Duncan & Flockhart to be far the best for this purpose, as it can be pushed well into the opening and thus there is no loss of fluid. On the other hand, the nozzle is very apt to leak, and I have sometimes found that the tube which I left nearly full has emptied itself into thin air.

Hewitt prefers to place a measured quantity of ethyl chloride in an open tube, which is then fixed to the lower end of the bag. As the glass tube is tipped up more and more of the fluid escapes into the bag and becomes vaporised.

For satisfactory results it is essential that the patient be properly prepared for a general anæsthetic. In cases of emergency this may be dispensed with, but the extra risk must be fully appreciated. The usual routine examination of heart, etc., should not be omitted. The position of the patient is not of great importance, and there is no doubt that ethyl chloride may be given to persons in the sitting posture with safety. Naturally for patients with weak hearts or any other marked disability the recumbent position would be preferred.

Many writers insist that it is necessary to insert some form of gag or prop between the teeth before commencing the administration. In cases where the operation is to be performed on the mouth or throat this is undoubtedly the case, as otherwise much of the short period of perfect anæsthesia may be lost in forcing the mouth open. In other cases, however, I have never inserted a prop, nor have I ever had occasion to regret the omission. As with all other anæsthetics it is essential to have a box-wood wedge at hand in case of emergency. The gag is likely to frighten or annoy the patient and I do not think that it is necessary.

The face-piece is placed over the patient's mouth and nose, and he is told to close his eyes and breath quietly. When the bag is fairly filled and his confidence is obtained a small quantity of ethyl chloride (about 3–4 c.c.) is spurted into the bag, the patient being previously warned not to "mind the rushing noise." In nervous patients there may be some holding of the breath or even struggling, but as a rule the breathing gradually becomes deeper and more regular, passing later into slight stertor. At the same time the face becomes somewhat flushed, the eyeballs tend to roll about and later become fixed. The corneal reflex now disappears and the patient may be considered to be well under the anæsthetic, and likely to remain so for about two minutes or possibly more. The behaviour of the pupils varies somewhat, but the rule is that they dilate as the anæsthesia progresses. In some cases, however, they remain contracted. The corneal reflex need not be abolished if only a very speedy operative procedure is contemplated. Another sign that presents is a considerable amount of spasm of the orbicular muscles and there is some danger that the unwary may mistake this for a corneal reflex long after the latter is abolished. Some spasm of the lower jaw and contraction of the sterno-mastoids is also present.

If the first spurt of ethyl chloride is not sufficient a second or even a third dose is given. In fact, the great advantage of this method is that the dose given is suited to each individual case. I frequently find that if the anæsthesia is just insufficient

a very small additional dose will quickly render it perfect. If there is any struggling, followed, as it often is, by specially deep and rapid breathing, there is danger of overdose. This is especially the case in children, who are apt to struggle owing to fright and then go very deeply under with unexpected rapidity. My advice is to watch the corneal reflex carefully and remove the face-piece as soon as it is abolished, if not before. If a deeper and longer anæsthesia is required it may be necessary to go beyond this point, but this should not be attempted by anyone who has not attained considerable experience in the use of the anæsthetic.

The dosage required varies greatly with the individual and the depth (and length) of anæsthesia required. For children up to twelve years of age 3–4 c.c. are usually sufficient, for adults 5–7 c.c., and for especially muscular or alcoholic subjects up to 10 c.c. I must admit that I sometimes find patients who for no obvious reason take a surprising amount of ethyl chloride and still remain but lightly under its influence.

Towards the close of the operation, especially if this has taken longer than expected (no infrequent occurrence), there may be some struggling and crying, but this is quite unconscious, and, after recovery, the patient remembers nothing of it. The recovery is usually rapid and quite uneventful. If delayed, sponging the face with cold water is recommended, but I have never had occasion to try it.

It is frequently said that the use of ethyl chloride is attended with much depression, vomiting, giddiness and headache. I am inclined to think that the after-effects are rather more severe than those of nitrous oxide, but that they have been considerably exaggerated. The patient can almost always walk home afterwards. When ethyl chloride is used as a preliminary to chloroform or ether I find that, in this hospital, we get very little vomiting, though I am not prepared to say that the ethyl chloride affects it in one way or the other. It is used much in operations on the nose and throat, which are frequently attended by swallowing of blood, which in itself causes vomiting. However, every anæsthetist is prepared to be blamed occasionally for what is really an effect of the operation.

Before leaving the details of the administration a word should be said as to the amount of air to be allowed. On this point there is some difference of opinion. Some advise a breath of air as soon as the breathing becomes regular and another just before the anæsthesia is complete. My own opinion is that better results are obtained if, starting with the bag full of air, all further supply of air is excluded. When this is done there is no cyanosis. But it is important that the bag should be well filled at the commencement. The bag I usually employ has strips of whalebone to keep it partly expanded. Another I use at an institution tends to collapse completely and some cyanosis is apt to result.

Ethyl chloride is an extremely useful agent for the rapid induction of anæsthesia before a prolonged operation. With its use the patient is usually unconscious after three or four breaths, and thus an extremely uncomfortable and distressing process is greatly curtailed. Early in my experience with ethyl chloride I was much impressed by what I was told by a medical friend of mine to whom I gave it preparatory to an appendicectomy. He took it decidedly badly, held his breath, struggled, and did not apparently lose consciousness for a long time. A few days later he said—"What a beautiful anæsthetic you gave me. I took about two breaths and went to sleep."

The sequence that I am most accustomed to use is ethyl chloride followed by chloroform. The procedure is quite simple. Ethyl chloride is given as described already until the breathing becomes automatic. A fairly concentrated vapour of chloroform is then administered by any method the anæsthetist prefers. In short, sufficient ethyl chloride is given to enable the patient to inhale quickly a vapour much stronger than he could respire if conscious. The net result is that the patient becomes unconcious almost at once and is ready for the operation a very few minutes later. Only one word of warning is necessary. In passing over rapidly to chloroform we sometimes get a condition where the breathing is quiet and the pupil widely dilated. It is not always easy to be sure whether this is due to the ethyl chloride or to an overdose of chloroform. Usually it is the effect of the chloroform in quieting the breathing while the dilatation of the pupil due to the ethyl chloride has not yet passed off. A rapid decision is necessary, and Barton points out that the condition of the jaw-muscles usually gives us the clue. If there is some spasm of the jaw the condition is due to the ethyl

chloride and the administration of the chloroform may be continued, when the pupil will soon be found to contract. I find that the condition of the sterno-mastoids usually gives the same information. However, when really in doubt the obviously safe procedure is to remove all anæsthetic at once.

Ethyl chloride also forms a good introduction to ether. This sequence may be given in various ways with the Clover inhaler or any of its modifications. One method is to set the pointer at "o" and spray the desired dose of ethyl chloride into the bag either direct or through the face-piece, the reservoir having been previously charged with ether as usual. The patient then inhales the ethyl chloride and the ether is given as soon as he is able to breathe it readily. Or ethyl chloride may be sprayed into the ether chamber, and then as soon as the patient is under its influence the ordinary quantity of ether is introduced in the usual manner. This is a simple but rather awkward method, as there is some liability for the patient to "come round" while the ether is being introduced. In using either of these methods it is important to introduce the least possible dose of ethyl chloride likely to be effective, as once in the inhaler that is to be used for the ether the whole dose given will be inspired. That is in itself a real disadvantage. Therefore I prefer the more clumsy method of giving ethyl chloride in a special inhaler as usual, and then at the right moment replacing it by a Clover, which is placed at hand ready charged with ether.

The modern tendency, however, is against the closed method of giving ether, and this fact alone has largely diminished the value of the ethyl chloride-ether sequence. No doubt it is possible to use ethyl chloride for the induction of an open ether anæsthesia, but there is sure to be some difficulty in getting the patient sufficiently under the open ether in time, and for this reason I do not think the sequence is one to be recommended for routine use.

Other sequences are also recommended. Barton speaks highly of a method by which he gives first C.E. mixture, then ethyl chloride and then more C.E. or chloroform alone. The method is slower and more complicated than the ethyl chloride-chloroform sequence and I do not quite appreciate its advantages. Again, others advise a combination of ethyl chloride with nitrous oxide. I look upon these two anæsthetics as playing very similar *rôles*, and any slight advantage in using them in combination scarcely seems to justify the loss of simplicity.

In the use of ethyl chloride for longer operations I have no personal experience, as I do not consider it a suitable anæsthetic for the purpose. Still, it is well to remember that it can be so used in case such an anæsthetic is urgently wanted and no other means are available. The simplest method to adopt is to get the patient well under in the ordinary way, and then as soon as the anæsthesia is passing off administer a further dose and so on. This involves a very careful watching of the respirations, the corneal reflex and the pupils, and it is by no means easy to avoid overdose on the one hand or return to consciousness on the other. At the best one gets an "up and down" result far removed from the level anæsthesia at which one should always aim.

Barton has devised a method especially suitable for longer operations on the throat and nose. A metal bottle containing the ethyl chloride is immersed in warm water, and the resulting gas is conducted through rubber tubing (which can be pinched to control its flow) to an opening on the inner side of a special gag.

On the Continent other and more elaborate forms of apparatus for the prolonged administration of ethyl chloride have been devised, but I have not heard that any of them have been generally used in this country.

It must be admitted, then, that in ethyl chloride we have an anæsthetic which is certainly very useful for some purposes. Its merits must be most nearly compared with those of nitrous oxide as both have very similar uses. First as to the question of safety. When first introduced ethyl chloride was apparently claimed to be that Utopian dream, "a perfectly safe anæsthetic." All anæsthetics are poisons or they would scarcely render the patient unconscious, and an overdose of any poison is dangerous. The safety of any anæsthetic depends not so much on whether an overdose will kill, as on whether it is likely to do so suddenly without giving an observant anæsthetist warning of the approach of danger. Nitrous oxide is very safe because the effects of an overdose are obvious before they are very dangerous. In this respect ethyl chloride is not quite so safe, but the signs of danger are not likely to be missed by a competent

observer. The general opinion is that ethyl chloride, in the matter of safety, is intermediate between nitrous oxide and ether, but much nearer the former. As to the actual mortality, the figures vary between one death in 2500 cases and one in 150,000: in other words, they are valueless. In this hospital ethyl chloride is given to between seven and eight hundred cases in the throat department yearly and I can find no record of any fatality. Some time ago Dr. Beresford Kingsford stated that it was given in 10,000 consecutive cases at the Central London Throat Hospital, with no accident except that artificial respiration had to be used twice.

A great advantage of ethyl chloride is its portability as compared to heavy gas cylinders. Again, one always has a haunting suspicion that the latter may be empty whereas the ethyl chloride can be seen in its glass tube. We have seen that the after-effects of ethyl chloride are rather more severe, but against that is to be placed the fact that we have an anæsthesia of longer duration. For work in a dental surgery where all the apparatus, including a nasal inhaler, is at hand, nitrous oxide is probably the better anæsthetic of the two. But for dental work at a patient's house ethyl chloride is more convenient, and I also prefer it for children, who are not always very good subjects for gas. Old people also usually take ethyl chloride well.

Naturally ethyl chloride is not suitable for all cases. In any case presenting some special difficulty or cause for anxiety I prefer to induce anæsthesia very gradually with ether or chloroform. Under this heading I would mention cases of serious glycosuria or albuminuria, uncompensated heart disease, empyema or large pleural effusion, and especially any case likely to show serious obstruction to the air-passages. Slight bronchial affections or even early pulmonary tuberculosis are not contra-indications.

For the removal of tonsils and adenoids in institutions where speed is of importance ethyl chloride is very suitable in spite of the above remarks about the air-passages. For work in a private house I prefer a carefully administered mixture of chloroform and ether (the proportion varying with the age of the child), because owing to the more gradual return to conciousness there is less likely to be screaming and crying immediately after the operation. Such crying is distressing to the patient's friends, who are frequently listening in an adjoining room and do not realise that the operation is finished.

In conclusion, a few words as to the physiological action of the drug. Observations on the patient are rather difficult and inconclusive, as with a drug acting for so short a time it is not easy to eliminate the effects of the emotional disturbance immediately preceding it. However, experiments on animals have been made by two independent colonial investigators—Dr. Webster and Dr. Embley. Without going into detail the results are as follows. Small doses increase the frequency and the depth of the respirations, and large doses diminish both. Small doses increase the blood-pressure, and large doses depress it chiefly, if not entirely, by vagus inhibition of the heart. It was found that to stop the heart in animals ethyl chloride had to be presented about nineteen times more concentrated than chloroform.

Anyone interested in this subject will find very full information and also references in Dr. McCardie's papers, in Mr. Barton's little book, 'A Guide to the Administration of Ethyl Chloride' (Lewis, 2*s*.), to which I am particularly indebted, and the last edition of Sir F. Hewitt's 'Anæsthetics.'

March 31st, 1913.

Pulsations in the Primitive Cardiac Tube.—In a case of abortion occurring six weeks after the last menstruation Armann secured an intact ovum as large as a hazelnut, of light grey colour and with well-developed chorionic villi. He opened the sac with scissors and observed distinct pulsations in the primitive heart. The embryo was 2·5 mm. long, and the yolk-sac was distended and as large as a lentil. The well-marked rhythm of the heart was noteworthy: 90 pulsations per minute were counted. The cardiac tube was as large as the head of a pin and no auricle or ventricle could be determined. The contractile movements continued for 15 minutes and then became less distinct, slower, more superficial, and about 5 minutes later they ceased. The specimen was placed in alcohol and measured and compared with the plates in Kollman's embryology. It corresponded in size to an embryo of about 14 days, which agreed with the history given by the patient.—*Journ. A.M.A.*, vol. lx, p. 695.

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